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VOLUME 28
1932

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

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JULY, 1932

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, Jan. 7, 1918, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

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Archives of Neurology and Psychiatry

VOLUME 28 JULY, 1932 NUMBER 1

CORTICOFUGAL AND ASSOCIATION FIBERS ARISING FROM THE CORTEX OF THE VERMIS OF THE CEREBELLUM

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This is a report of a study of the secondary degeneration of the nerve fibers resulting from lesions in the cerebellum of dogs that had been used in a previously reported experimental study of the cerebellar control of the vocal cords.[1]

MATERIAL

The operations were performed in the Physiological Laboratories of the University of Amsterdam with the help of Professor G. van Rijnberk. Physiologic studies were made on the dogs after operation. The majority were killed on the eighteenth day. The brains were then examined grossly and prepared in the Neurological Laboratory of Professor B. Brouwer of the Binnen Gasthuis for study by the Marchi method, and complete serial sections were cut. The operative lesions were studied microscopically. A complete report of the physiologic studies and the extent of the operative lesions, together with conclusions, has been published.[1] In summary, these findings were that: "Lesions of any part of the vermis, of the roof nuclei, or the decussation between these nuclei do not produce changes in the function of the vocal cords of dogs, but that lesions in those tracts which connect the cerebellum with the medullary nuclei do produce motor disturbances of an ataxic type. Therefore the cerebellum appears to have some control over the motor activities of the vocal cords though this control has not been localized in any particular part of the cerebellum so far studied."

A study of the secondary tracts of degeneration was made under the direction of Professor B. Brouwer and Professor C. U. Ariëns Kappers of the Central Institute of Brain Research of Amsterdam.

It is not my aim in this study to determine the specific tracts that were responsible for the disturbance in the motor function of the vocal cords. As was reported in the first paper, the area that was injured in the dogs that showed such disturbances was the area of Meynert's "innere Abteilung des Kleinhirstieles," or the "IAK," which includes the tractus cerebellonuclearis, composed of ascending and descending fibers connecting the various nuclei of the medulla and cerebellum. This material gave opportunity for the study of certain outgoing fibers

From the Neurological Laboratories of the Binnen Gasthuis and the Central Institute of Brain Research, Amsterdam, Holland.

1. Bender, Lauretta: The Cerebellar Control of the Vocal Organ, Arch. Neurol. & Psychiat. **19**:796 (May) 1928.

and systems of fibers the cells of which originated in the vermis, especially in its most anterior part. No effort is made at functional correlations, but this paper reports merely the anatomic relations of these fibers.

The classic English work on the course of the fibers arising from the cortex of the cerebellum is that of Clarke and Horsley.[2] They concluded that no fibers issuing from the cortex of the cerebellum enter any of the peduncles, but all end in one or another of the nuclei. They demonstrated also the presence of intracerebellar association tracts, the importance of which has not been sufficiently emphasized. They seemed to consider the nucleus of Deiters as a "central" cerebellar nucleus and the fibers that pass to it in the "IAK" as not a part of the cerebellar peduncle, although they did not state this in the text. A study of their protocols shows that degeneration of such fibers often occurred. They reviewed the literature preceding their work and reported that Marchi, Ferrier and Turner, Russel, Münzer and Wiener did not find any fibers arising in the cerebellar cortex which passed beyond the cerebellar nuclei, although Biedl, Thomas, Ramón y Cajal and Probst claimed that such fibers existed. The most extensive work was that of Thomas,[3] who performed forty-two experimental lesions on the cerebellum in dogs and studied them by the Marchi technic. He concluded that lesions of the cerebellar cortex produced degeneration in fibers passing to all of the cerebellar nuclei, to the vestibular nuclei, to the pons and to the lateral and cuneate nuclei of the bulb.

Fusé[4] reviewed the literature up to 1912, and made some contributions of his own by a thorough study of the "innere Abteilung des Kleinhirnstieles." He quoted the work of Teljatnik, Probst, Orestano, Lewandowsky, van Gehuchten and others who traced fibers from the cerebellar cortex to other parts of the central nervous system. The work of these men will be considered more in detail as the individual tracts are discussed. Cajal,[5] by his special methods, thought that he was able to follow the axons of Purkinje cells into the inferior peduncle after they had given off collaterals to the cortical folia and to the roof nuclei and had decussated at the level of the vermis. His preparations were not adequate to follow these fibers to their destination. This work was done on new-born and fetal rats.

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2. Clarke, R. H., and Horsley, V.: On the Intrinsic Fibers of the Cerebellum, Its Nuclei and Efferent Tracts, Brain **28**:13, 1905.

3. Thomas, André: Le cervelet, Paris, G. Steinheil, 1897.

4. Fusé, G.: Die innere Abteilung des Kleinhirnstiels (Meynert, IAK.) und der Deitersche Kern, Arb. a. d. hirnanat. Inst. in Zürich **6**:29, 1912.

5. Ramón y Cajal, S.: Beiträge zum Studien der Medulla oblongata, des Kleinhirns, und des Ursprungs der Gehirnnerven, Leipzig, Johann Ambrosius Barth, 1896.

The experiments here reported were so planned that lesions were produced in unusual places, since an effort was made in most cases to produce lesions in the superior vermis of cephalic lobe of the cerebellum by passing through the fourth ventricle. The Marchi preparations were satisfactory, and the demonstrated degenerations were in some cases so unusual as to throw unexpected light on the course of the efferent tracts of the cerebellum.

TECHNIC

The work was done in Professor Brouwer's laboratory, and the technic was as follows:

1. Fix whole brains by suspending them in a 10 per cent solution of commercial formaldehyde for twenty-four hours.

2. Wash in running tap water for one-half hour.

3. Cut in pieces not to exceed from 2 to 3 mm. in thickness and place in Müller's solution for from three to five days.

4. Leave for four days in the dark in a mixture of two parts of Müller's solution and one part of 1 per cent osmic acid solution.

5. Place in a mixture of one part of Müller's solution and one part of 1 per cent osmic acid solution in the dark for twenty-six days. The blocks should be turned every day but kept immersed in the solution. The dishes should be kept covered and the edges of the covers protected with petrolatum to keep out the air. The blocks should be handled with camel hair brushes. A piece of filter paper should be kept beneath the blocks on the bottom of the container, and the blocks should not be allowed to touch one another.

6. Wash in several changes of distilled water, dehydrate through graded alcohol, embed in celloidin, section at 40 microns, dehydrate through the alcohols, carbol-xylene and xylene, and mount in balsam.

For complete physiologic and anatomic protocols of these experimental animals, reference should be made to the earlier paper.[1] In this paper only those fibers or tracts that arise from the cortex of the vermis will be followed. In no instance was there any evidence to suggest retrograde degeneration, especially as an effort was made to follow the degenerated fibers through serial sections from the cortical lesions; therefore the conclusions have been based, rightly or wrongly, on the assumption that there was no retrograde degeneration.

PROCTOCOLS

Dog 23 (fig. 1).—The operative injury included a lesion in the cortex of the tuber and a more superficial one in the cortex of the pyramis. The secondary fiber degeneration involved: (1) fibers that passed from one part of the cerebellar cortex to another part (cerebellar association fibers); (2) fibers that passed from the cerebellar cortex to the central cerebellar nuclei (short corticofugal fibers), and (3) fibers that passed from the cerebellar cortex out to the brain stem (long corticofugal fibers). In reference to these groups, the terms short and long corticofugal fibers will be used with the connotation already given.

Of the short corticofugal fibers that arose from the cortex of the tuber and pyramis, many ended in the two roof nuclei, a few in the globoid nuclei and a very few in the emboliform nuclei. None entered the dentate nuclei.

The long corticofugal fibers in this animal constituted two distinctly different bundles. One included fibers that passed directly to the nuclei of Deiters and of Bechterew in a position medial to the restiform body, as the "fibrae perforantes" or perforating fibers of the cerebellonuclear tract. The other included fibers that passed through and between the central nuclei to the root of the lingula, where some of them decussated ventral to the central nuclei, hooked about the brachium conjunctivum, ran in the tract of Russel and ended after descending a short distance in the nucleus of the descending root of the vestibular nerve. This was the tract that I shall designate as the bundle of ventral decussation of the cerebellum, or more briefly, the ventral bundle (fig. 9). In this brain, in which the lesion was small and involved only portions of the inferior vermis (the caudal

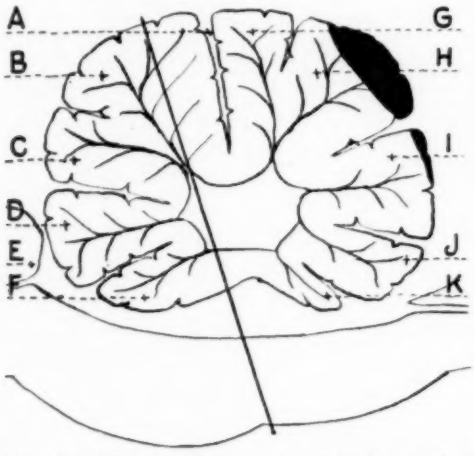

Fig. 1 (dog 23).—A schematic diagram through the cerebellum drawn in the mesial plane to show the extent of the lesion. The diagonal lines in the schematic diagram indicate the plane on which the serial sections were cut. In this figure and in figures 2, 4, 6 and 8, *A* indicates s. primarius (fissura primara); *B*, monticulus (lobulus IV vermalis); *C*, declive (lobulus III vermalis); *D*, l. centralis (lobulus II vermalis); *E*, c.quad.ant.; *F*, lingula (lobulus I vermalis); *G*, l. simplex (lobus simplex); *H*, tuber (lobus C_2 vermalis); *I*, pyramis (lobus C_1 vermalis; J, uvula (lobus B vermalis); *K*, nodules (lobus A vermalis). In agreement with the first paper, Bolk's terminology is used in this paper also, except that occasionally Ingvar's concepts are illustrated with his special terms, but when this occurs it is indicated. Recently Riley, H. A. (Lobules of Mammalian Cerebellum and Cerebellar Nomenclature, Arch. Neurol. & Psychiat. **24**:227 [Aug.] 1930) suggested a simplified revision of Bolk's terminology which might well have been adopted in this paper had it not been completed and the first paper published prior to the publication of Riley's recommendation. However, this revised terminology is indicated in the foregoing in the parentheses.

lobe of the cerebellum), the number of degenerated fibers was small; no degeneration was found in ascending fibers, and all ended after a short course in the adjacent parts of the nucleus of the descending root of the vestibular nerve.

The association fibers that arose from the cortex of the pyramis were seen to be connected with the other folia of: the pyramis, the tuber, a very few with the uvula and nodules, and a few apparently with the flocculi and the paramedial lobules. From the tuber, association fibers were followed to the other parts of the tuber, the pyramis, both paramedial lobules and the lobulus simplex, including its most lateral extensions. The degenerated association fibers were much more numerous in the upper part of the caudal lobule than in the lower part.

Summary.—Even in this animal, in which there was a relatively limited and superficial lesion in the cortex of only the tuber and pyramis, degenerated fibers could be followed into all adjacent parts of the vermis as association fibers; into all the central cerebellar nuclei as the short corticofugal fibers ending in the adjacent regions of the floor

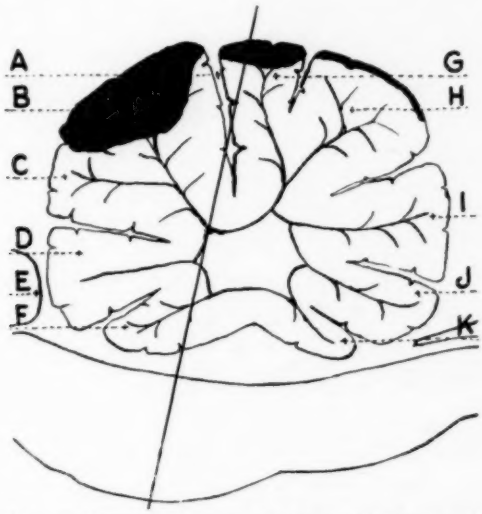

Fig. 2 (dog 11).—Schematic diagram of the cerebellum in the mesial plane to show the extent of the lesion.

of the fourth ventricle, and also to some degree in the bundle of the ventral descussations to more remote parts of the brain stem, at least in the caudal direction.

Dog 2 (fig. 2).—The operative injury included a deep lesion in the culmen, a very slight one in the declive and a superficial one in the lobulus simplex in the midline, and one folium of the left paramedial lobule.

Degenerated fibers were again seen to follow the course of the short and long corticofugal and the association tracts.

The short corticofugal fibers ended in the roof nuclei, the globoid nuclei and the emboliform nuclei, but a considerably greater number ended in the more lateral nuclei than in the case of dog 23 in which the lesion was limited to the more inferior parts of the vermis. In addition, a distinct tract of fibers passed beyond the ventral nuclei dorsal to the dentate nuclei to end in its most lateral, dorsal and oral portion, which is phylogenetically oldest and closely related to the central nuclei.

The long corticofugal tracts were seen to follow four distinct courses:

1. Fibers passed through and beyond the central nuclei without decussating as the perforating fibers or cerebellonuclear tract. Of these a large bundle of fine fibers (fig. 3 *B*) passed to the nuclei of Deiters and Bechterew, running between the roof nuclei and the globoid or emboliform nuclei, although some came through the globoid nuclei themselves and might be misinterpreted as arising there. These took a lateral course in the inferior peduncles. Other coarser fibers (fig. 3 *C*)

Fig. 3.—A vertical incision through the inferior cerebellar peduncle of dog 11. To the right is seen the fourth ventrical containing the choroid plexus and parts of the cortex of the lingula, *A*. The degenerated fibers are seen as two distinct bundles. There is a lateral bundle of fine fibers at *B*. These pass to the nuclei of Deiters and Bechterew. There is also a medial bundle of coarse fibers at *C*. These end in the dorsal nucleus of the eighth nerve, in the mesial and ventral vestibular nuclei mass or in the reticular formation. (Marchi preparation.)

lay medial to these. They were found in greater numbers in the more caudal sections. They ended either in the dorsal nucleus of the eighth nerve, in the

medial and ventral nuclei of the vestibular nerve or in the reticular formation. They seemed to take a course similar to the striae acusticae and were most numerous at that level. From here their course was to the posterior longitudinal bundle, to the reticular formation or to the descending tract of Deiters' nucleus.

2. A fair number of fibers passed from the cortex to enter the bundle of the ventral decussation. They were coarse enough to be followed through the nuclear mass easily. After decussating they hooked over the brachium conjunctivum, took the position of the tract of Russel and ended at all levels in the spinal vestibular nucleus which they accompanied downward.

3. There were also a few degenerated fibers decussating in a position dorsal to the central nuclei, which will be called the bundle of the dorsal decussation (fig. 10) or, briefly, the dorsal bundle. Two parts could be recognized in this tract. A longer part arose from the base of the lingula ventral to the roof nuclei, decussated and passed to the restiform body by a circuitous route, as shown in figure 10, *2*. A shorter group arose from the other parts of the superior vermis to reach the restiform body directly (fig. 10, *1*).

4. Degenerated fibers to the brachium conjunctivum were seen passing through and between the roof nuclei. They ascended in the brachium conjunctivum to the midbrain. Many of them decussated and ended in the red nucleus, while others continued as the cerebellar tegmental tract to end about the large nerve cells of the ipsolateral mesencephalic nucleus of the fifth nerve (proprioceptive).

Degenerated association fibers were seen in all parts of the culmen, declive, tuber and pyramis, and the lobulus simplex to its most lateral extensions. A moderate amount of degeneration was seen in the lobulus centralis and in all parts of the paramedial lobules and both flocculi. A slight amount was seen in the uvula. There was none in the lingula, nodulus or hemispheres.

Summary.—In this experimental animal the injury was limited to the culmen, except for a slight involvement of adjacent parts of the vermis. Thus, the injury was in a more superior part of the vermis than that in the previous animal. The degenerated fibers were again seen in adjacent parts of the vermis as association fibers to the central cerebellar nuclear mass, as short corticofugal fibers, but with a greater distribution, to the more lateral nuclei and into the adjacent parts of the floor of the fourth ventricle, as perforating fibers, and to the more distant parts of the brain stem in both the bundles of the ventral and the dorsal decussation and in lesser number in the brachium conjunctivum to the midbrain.

Dog 17 (fig. 4).—The operative injury included slight lesions of the ventral surface of the uvula and nodulus, a more severe lesion of the lingula and its base, and slight injury to the caudal end of the floor of the fourth ventricle, the medial surface of the left inferior peduncle and the posterior quadrigeminal body.

Association fibers were degenerated in all parts of the vermis, the lobulus simplex, the paramedial lobules and the flocculi.

Among the long corticofugal or efferent fibers that showed degeneration were the perforating fibers. Large bundles of fine fibers passed from the roof nuclei through the inferior peduncles to the more dorsal vestibular nuclei, where they ended. Coarser black fibers passed to the more ventral portion of the vestibular

nuclear mass, the dorsal nucleus of the eighth nerve and the reticular formation. To this region fibers from the tract of Russel were also contributed.

Broad bands of degenerated fibers arose from the lingula to enter the bundle of the ventral decussation (fig. 5). They did not all decussate. The tract then hooked over the brachium conjunctivum and took its position as the tract of Russel. The ascending part soon joined the fibers from the bundle of the dorsal decussation, the two being separated by the restiform body until it disappeared. They then passed at various levels as external arcuate fibers which could be traced in part to the nuclei of the raphe in the midbrain. The descending branches from their position in the tract of Russel sent off fibers to the reticular formation, the descending tract of Deiters nucleus and nuclei in the region of the posterior longitudinal bundle. Most of the fibers of this tract terminated at the various levels in the spinal vestibular nucleus, which they followed closely. Along its course fibers were given off to join the external arcuate fibers from the restiform body.

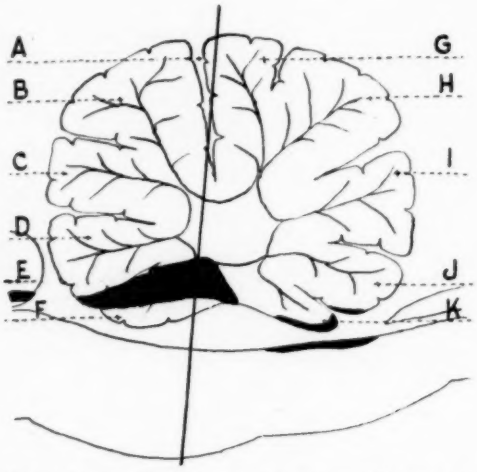

Fig. 4 (dog 17).—Schematic diagram of the cerebellum drawn in the mesial plane showing the extent of the lesion.

Degenerated fibers of the dorsal bundle passed from the lingula through the fibers of the ventral bundle between the roof nuclei. Here they decussated and then curved over the nuclei and down between the dentate and emboliform nuclei near the restiform body (figs. 5 and 10). They divided into ascending and descending bundles to encircle ultimately and cross the brain stem as external arcuate fibers. Those of the upper levels passed between the medial lemniscus and the pons and ended in the nuclei of the raphe. In the medulla they passed around or through the pyramids and ended in the nuclei of the raphe. Others passed to the inferior olive. There was moderate degeneration of the fibers in the brachium conjunctivum, which decussated and ended in the red nucleus of the opposite side.

Summary.—This animal presented the strongest experimental evidence for the thesis of this paper. Though the injury was pretty well limited to the lingula, degenerated association fibers could be followed

to all parts of the vermis, even to the flocculi; the short corticofugal fibers could be followed to the central cerebellar nuclei, and the long corticofugal fibers could be followed as the perforating fibers to the adjacent parts of the floor of the fourth ventricle, to many adjacent and distant parts of the brain stem through the numerous ramifications of the bundles of the ventral and dorsal decussations, and to a lesser extent through the brachium conjunctivum to the midbrain.

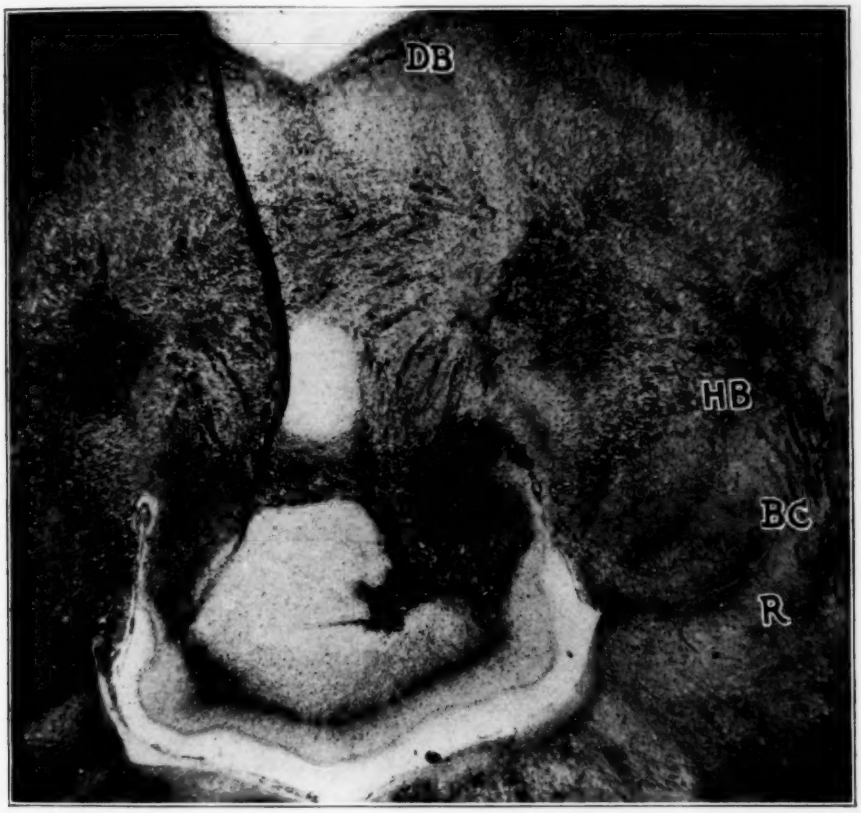

Fig. 5 (dog 17).—Vertical section through the cerebellum and brain stem showing the cortex of the lingula projecting into the fourth ventricle in the center, into the two superior peduncles on either side and into the central cerebellar nuclei above. In the lingula the operative lesion is to be seen as an irregular black mass a little to the right of the center. Black degenerated fibers are seen in the tracts of both the ventral and the dorsal bundles. Some fibers of both bundles decussate, and some do not. Both crossed and uncrossed fibers of the ventral bundles pass over the ventral brachium conjunctivum (*B C*) as the hooked bundle (*H B*) to reach the tract of Russel (*R*). Only a few degenerated fibers of the dorsal decussation can be recognized here. They have passed up through the center between the nuclei and are seen running outward and upward at the top of the photograph, just below the small v-shaped clearance that is underlying the cortex.

A similar lesion was produced in dog 25 (fig. 13 in my previous study [1]), except that it extended into the lobulus centralis, but this dog lived only eleven days and the picture obtained by the Marchi method was a little less clear than the pictures in the other animals. However, the findings were essentially the same as those in dog 17.

Dog 18 (fig. 6).—The operative injury passed transversely through the cerebellum. The pyramis was largely destroyed, while the adjacent surfaces of the uvula and paramedial lobules were slightly injured. The lesions also involved both roof nuclei and the right globoid and emboliform nuclei. The forward end of the lesion passed into the lobulus centralis. Thus, in this animal were combined the lesions of the former experimental animals, with the addition of a lesion in the central nuclei. Of course there was extensive degeneration of fibers running to

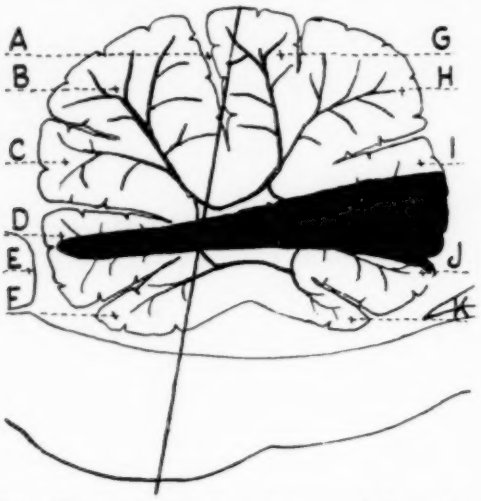

Fig. 6 (dog 18).—Schematic drawing of the cerebellum showing the extent of the lesion.

various parts of the cortex, both because numerous association fibers were cut and because of injury to corticopetal fibers. These are not of concern here. Interest in this study lies in the fact that the extensively degenerated corticofugal fibers to the brain stem followed the same course as those in animals in which the cortex alone was injured. Apparently no new pathways had degenerated by reason of destruction of the central nuclei, but the degeneration was greater in amount in the same paths that were involved in pure cortical lesions. Figure 7 shows the extensive degeneration of the ventral bundle as it capped the brachium conjunctivum. This is the tract of Russel of the older literature, and it is easy to see how it was assumed that this arose from the central nuclei, which was the usual seat of experimental lesions, since the superior parts of the cortex were difficult to reach. In this animal the brachium conjunctivum showed more degenerated fibers than in the previous ones, especially on the right side where the lesion was most extensive in the nuclei, but, even so, the greater part of the brachium conjunctivum was free from degeneration, as would be expected since it arose for the most part in the dentate nucleus, which was injured.

Dog 7.—As shown in figure 8, the extent of the operative lesion appears to be not unlike that in dog 18, but more extensive. Cross-sections, however, show that the lesion passed through the center of the cerebellum in such a way as to separate the two roof nuclei in the midline without apparently injuring them to any degree, but neatly cutting through the point of decussation of both the ventral and dorsal bundles; and thus there was produced a very extensive degeneration of all the fibers of both of these systems, and the distribution was found to be the same as those in the previously described cases.

Fig. 7 (dog 18).—A vertical section through the superior cerebellar peduncle. On the left is seen the restiform body (*R B*), on the right the lingula (*L*) and between them the brachium conjunctivum (*B C*). The dark fibers hooking over the brachium conjunctivum are those of the hooked bundle (*H B*) that are passing to the tract of Russel (*R*). (Marchi preparation.)

Dogs 10 and 14 (figs. 13 and 16 in my previous work [1]).—These animals also had severe lesions through both the inferior and superior parts of the vermis and the central nuclei, and to some extent at various points in the brain stem. The degenerated fibers were extensive but could be seen to follow the same course as in the other animals and further confirmed the evidence already obtained.

SUMMARY OF THE LONG CORTICOFUGAL FIBERS OF THE CEREBELLUM

1. *Perforating Fibers.*—These fibers may arise from the cerebellar cortex, at least of the anterior lobe. Lesions of the posterior lobe alone did not produce any degeneration in them. They are more numerous, however, if the roof nuclei are also injured, but this may be due to the fact that in this way more of them are injured in their course. There is no evidence that any of them decussate. They may be divided into two groups: (1) a large bundle of fine fibers that pass chiefly between the roof and globoid nuclei, but also through them, and appear to end in the nuclei of Deiters and Bechterew; (2) coarser, darker fibers that pass close to the wall of the fourth ventricle and are

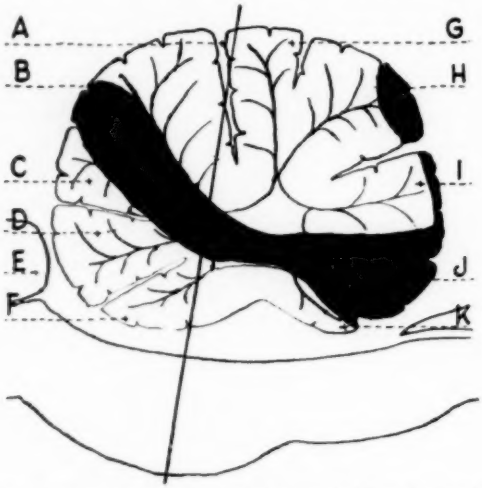

Fig. 8 (dog 7).—Schematic diagram of the cerebellum drawn in the mesial plane to show the extent of the lesion.

more abundant in the more caudal levels. These appear to end in the dorsal vestibular nucleus, the reticular formation and the more ventral portions of the vestibular nucleus. Some of them accompany the striae acusticae, but most of them turn back again.

The fibers traveling in the brachium conjunctivum reach this body at the same level as the perforating fibers, but are distinguished from them by the fact that they are coarse and darkly stained and tend to cross the fine perforating fibers to take their course in the superior peduncle where they ascend.

The course of the perforating fibers in the reticular formation will be considered separately, also those of the brachium conjunctivum.

The perforating fibers or fibrae perforantes are the fibers that were so carefully studied by Clarke and Horsley.[2] They claimed that lesions

of the cerebellar cortex produced degeneration of fibers to the cerebellar nuclei, which according to them seemed also to include the vestibular nuclei. Lesions of the central nuclei were reported to produce a degeneration of a large number of such fibers. These were the only efferent tracts that Clarke and Horsley recognized. However, in no instance did their lesions involve the lingula. Lewandowsky [6] described fibers passing from the cerebellum through the internal portion at the restiform body to Deiters' nucleus and scattering about the adjacent reticular formation. Edinger [7] also described fibers that arise in the roof nuclei, the globoid nuclei and the cortex of the vermis and pass through the IAK to the nuclei of the trigeminal, acoustic and vagus nerves, the posterior longitudinal bundle and the nuclei of Deiters and Bechterew. Cajal,[5] Klimoff [8] and Probst [9] described them in the same way. Similar efferent fibers are described by Cramer,[10] Bechterew [11] and Isao Yoshida.[12] The question as to whether the descending tract of Deiters' nucleus receives any fibers from the cerebellar cortex is discussed by Fusé,[4] who quoted all of the literature. He said that the question is answered in the affirmative by Probst, Marchi and Thomas, and in the negative by Lewandosky and Russel.

2. *The Bundle of the Ventral Decussation.*—The ventral bundle (fig. 9) is a corticofugal system that arises at least in part from the cortex of the vermis and especially from the lobus anterior. The fibers pass through the roof nuclei, where they may be joined by other fibers arising there. They pass to the base of the lingula where they are joined by many fibers arising from its cortex. Many decussate, but some do not. They then pass ventrally, many of them through the anterior medullary velum, and hook about the brachium conjunc-

6. Lewandowsky, M.: Untersuchungen ueber die Leitungsbahnen des Truncus cerebri; Neurologischen Arbeiten von O. Vogt, Jena, Gustav Fischer, 1904, series 2, vol. 1.

7. Edinger, L.: Bau der nervösen Zentralorgane, Leipzig, F. C. W. Vogel, 1911.

8. Klimoff, J.: Ueber die Leitungsbahnen des Kleinhirns, Arch. f. Anat. u. Entwcklingsgesch. **27**:11, 1899.

9. Probst, M.: Zur Anatomie und Physiologie des Kleinhirns, Arch. f. Psychiat. **35**:692, 1902.

10. Cramer, A.: Beiträge zur feineren Anatomie der Medulla oblongata und der Brucke mit besondere Berücksichtigung der 3-12 Stirnnerven, Jena, Fischer, 1894.

11. Bechterew, V.: Ueber die innere Abteilung des Strickkorpers und der achten Hirnnerven, Neurol. Centralbl. **4**:145, 1885.

12. Yoshida, Isao: Ueber die Nissl-Degeneration nach Kleinhirnläsionen: I. Ueber die Nissl-Degeneration in der Vestibularis-Endkernen und anderswo nach Kleinhirnläsionen, Okayama-Igakkwai-Zasshi (Mitteilung der Medizinischen Fakultät zu Akayam) no. 415 (Aug.), no. 416 (Sept.) 1924.

tivum, where they cover its surface like a cap and divide into an ascending and descending portion.

The fibers of the ascending portion follow the surface of the brachium conjunctivum and then follow an arcuate course to end in a mesial position. At higher levels, above the place that the restiform body is seen, the ascending fibers of the ventral bundle are joined by fibers from the dorsal bundle. Degeneration was observed in the

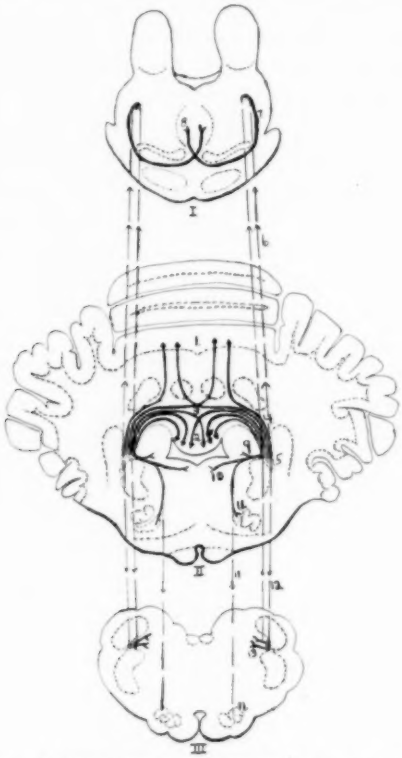

Fig. 9.—Diagrams of three levels of the central nervous system of the dog. I is at the level of the anterior quadrigeminal bodies. II is at the level of the cerebellar peduncles, showing the cortex of the vermis on the upper surface (declive or culmen of monticulus) and in the fourth ventricle (lingula) and also showing the central cerebellar nuclei. III is through the medulla. Fibers of the bundle of the ventral decussation arise from the cortex of the vermis, from the declive or culmen (1) and from the lingula (2). Some pass through the ventral decussation at (3). Both crossed and uncrossed fibers hook over the brachium conjunctivum at (4), forming the hooked bundle, and enter the tract of Russel (5). Here some fibers ascend (6) to the midbrain, where in the form of external arcuate fibers (7) they cross the midline and end in the nucleus of the raphe (8). Others pass to the reticular formation (9); others join the posterior longitudinal bundle (10), and others enter the descending tract of Deiters' nucleus (11). The major number of the fibers (12) descend along the descending vestibular nucleus (13) and end in it at all levels.

external arcuate fibers, many of which could be traced as far as the nucleus of the raphe, although many disappeared before this central position was reached. These were traced as far headward as the posterior quadrigeminal body. It is possible that these fibers are derived only from the dorsal bundle, since they were present only when the degeneration of this tract was most prominent.

The descending portion takes the course of the tract of Russel, following the spinal vestibular nucleus throughout its length and ending in it. At all levels, fibers pass into the reticular formation, the posterior longitudinal bundle and the descending tract of Deiters' nucleus.

When the cortex of the posterior lobe or of the inferior vermis alone is injured, few long corticofugal fibers are degenerated, and these end nearby in the upper parts of the vestibular nuclei. But when the anterior vermis and especially the lingula are injured, numerous long fibers are degenerated. These may be traced to the full extent of the spinal vestibular nucleus. Injury of the roof nuclei produces a degeneration of a very large number of such fibers. However, it remains to be proved that these actually arise from the nuclei, as all of the fibers from the anterior lobules pass through or near these nuclei. Degeneration appears bilaterally equal even when the injury is unilateral, indicating that about half the fibers decussate.

3. *The Bundle of the Dorsal Decussation.*—The fibers of the dorsal bundle arise mainly in the cortex of the anterior lobe, pass to the midline and decussate, and then pass to the region of the restiform body (fig. 10). Because of the different anatomic relations of the four lobules of the anterior lobe, owing to the long axis of the lingula being at right angles to that of the monticulus, etc., the fibers of the different lobes take different courses. Those from the lingula pass dorsally, penetrating the ventral bundle in the midline. Here some decussate and pass over the dorsal surface of the nuclear masses. They are then joined by fibers from the lobulus centralis, monticulus and declive. These arise from the base of the lobules, cross in the midline and pass laterally in broad bands ventral to the nuclear mass and converge to join the restiform body. From here they may follow either an ascending or descending course, and in this they are joined by fibers from the ventral bundle. Fibers derived from both dorsal and ventral bundles leave this position at all levels as arcuate fibers. At the level of the pons they pass between it and the mesial lemniscus to reach the nucleus of the raphe of the midbrain. At the level of the medulla they pass around or through the pyramids to reach the nucleus of the raphe of this level. Of the descending fibers, some end in the inferior line. In all cases many degenerated fibers disappeared before they reached the nuclei of the raphe. A much greater proportion of degen-

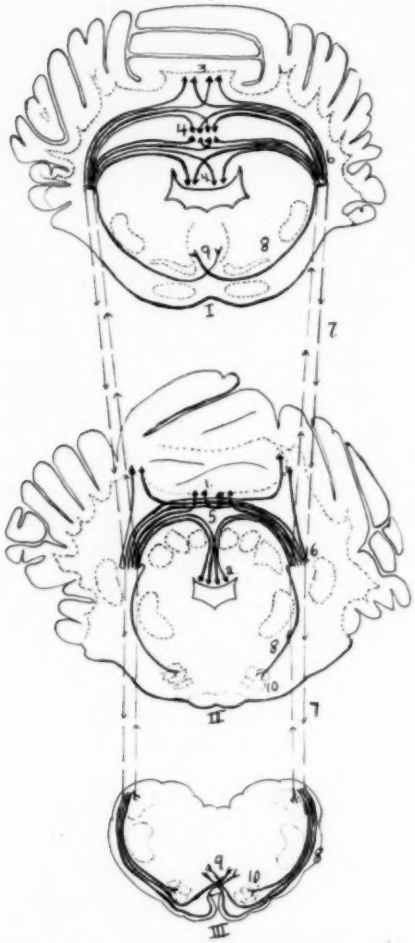

Fig. 10.—Diagram of three levels of the central nervous system of the dog. I is high in the pons and shows the anterior lobe of the cerebellum with the declive on top and the lobulus centralis projecting into the fourth ventricle. II is through the pons at a somewhat lower level, showing the culmen on top and the lingula projecting into the fourth ventricle and the central cerebellar nuclei. III is through the medulla. The fibers of the bundle of the dorsal decussation arise from the vermis in all its parts [from the culmen (1), lingula (2), declive (3) and the lobulus centralis (4)] and pass to the decussations (5). Those in the lingula must pass through the roof nuclei to reach this position. Some of the fibers do not decussate, but all lie above the roof nuclei, and then as a well defined bundle they pass down along the lateral surface of the restiform body (6). From here they either ascend or descend (7) and pass as external arcuate fibers (8) to end in the nuclei of the raphe (9) or the olives (10) at the various levels.

erated fibers was found to reach their central position when the lingula was injured than when the lesion involved other portions of the vermis.

In addition to the arcuate fibers, there are many that can be seen to end in the central gray matter. These are derived from the ventral bundle by way of the tract of Russel and from the perforating fibers. Some of them end in the gray matter accompanying the posterior longitudinal bundle, some in the gray matter of the reticular formations and some in the vestibular nuclei. Others accompany the descending tract of Deiters' nucleus and leave it at all levels, passing toward the inferior olive.

The tract that is here called the ventral bundle of the cerebellum has been variously called the "hooked bundle" of Russel, the "faisceau en crochet" or "faisceau cerebello-bulbaire" of André Thomas, the "tractus uncinatus" of Lewandowsky and the "Hackenbündel" of Probst. The literature on it has recently been reviewed by Mussen,[13] who accredited its discovery to Russel[14] in 1894. Russel ablated the lateral cerebellar lobes in dogs and found degeneration of a hooked-like tract which capped the dorsal region of the opposite superior cerebellar peduncle. However, he gave no details as to the origin of the fibers. Mussen also quoted Probst, Lewandowsky, van Gehuchten, André Thomas and Marburg, who all concluded that these fibers arise in the medial cerebellar nuclei, decussate beneath them and hook over the superior cerebellar peduncle. From here there is considerable disagreement as to their course. Probst[9] claimed that they turn ventrally and that some end in: Deiters' nucleus, Burdach's nucleus, the reticular formation, the inferior olive and the nucleus of the sixth nerve. But Probst described another set of fibers that arise from the cortex of the vermis and would seem to correspond closely with the dorsal bundle here described, although he did not recognize it as such. He claimed that they pass through the pontile peduncle to the pontile gray matter.

Edinger[7] is the only writer who makes a distinction between the ventral and dorsal cerebellar bundles. He recognized dorsal, medial and ventral groups of decussating fibers. He claimed that these all arise in the roof nuclei and stated that this dorsal bundle corresponds to the "Hackenbündel," and that the fibers end in various motor nuclei of the spinal cord and in Deiters' nucleus. They follow the descending root of the eighth nerve to the cervical level of the cord. Some, he said, take the form of external arcuate fibers to the nuclei of the raphe. He called this the "tractus cerebello-tegmentalis bulbi." The fibers that

13. Mussen, Aubrey, T.: Experimental Investigation on the Cerebellum, Brain **50**:313 1927.

14. Russel, J. S. Risien: Degeneration Consequent on Lesions in the Cerebellum, Brit. M. J. **2**:640, 1894.

decussate in the medial group he called the "tractus cerebello-tegmentalis pontis." These are in close relation to the pontile fibers and end in the reticular formation and the raphe. The ventral group he believed to be a commissure between the flocculi.

The work of Lewandowsky,[6] who used the Marchi method on experimental lesions in the dog, will be reviewed somewhat fully, since it is similar to my own both in the approach and in the conclusions. He found fibers that pass through the restiform body and the brachium pontis as the external arcuate fibers to end in the nucleus of the raphe. Other fibers, the perforating fibers (fibrae perforantes), pass through the internal part of the restiform body to Deiters' nucleus. Fibers of the tract of Russel (tractus uncinatus) showed bilateral degeneration in a lesion of one half of the cerebellum, including the roof nuclei. These fibers crossed in the decussation of the vermis, which according to his illustrations corresponds to my ventral bundle. These then hooked around the brachium conjunctivum and took a position between it and the lateral lemniscus. From there some fibers passed to the nuclei of Deiters and Bechterew, while some descended to the root of the acoustic nerve and the tractus solitarius. Others ascended as Thomas' retropeduncular tract to the nucleus of the lateral lemniscus.

Van Gehuchten [15] studied Marchi degeneration in rabbits, and came to similar conclusions, except that he recognized no cerebellofugal fibers in the restiform body and no decussation of the peduncular fibers in the cerebellum, but traced a portion of the "tractus uncinatus" into Deiters' and Bechterew's nuclei, the descending tract of Deiters' nucleus, the reticular formation and the cervical cord. He identified this with Edinger's cerebellar nuclear tract.

Bechterew [16] studied the tract of Russel and claimed that it arose chiefly from the dentate nucleus, but partly from the central nuclei, and he specifically stated that lesions of the cortex of the lateral hemispheres and of the vermis do not give rise to degeneration in these fibers. Bechterew [11] also described in the human fetus fibers that run between the cerebellum and the superior olive, crossing between the roof nuclei and descending in the restiform body. Winkler [17] believed that this tract arises partly in the roof nuclei and partly in the dentate nuclei, and after decussating passes to the vestibular nuclei. He described a bifurcation in Dieters' nucleus, a portion following the

15. van Gehuchten, A.: Les pédoncules cérébelleux supérieurs, Névraxe **7**:29, 1905; Le corps restiforme et les connexions bulbo cérébelleuses, ibid. **7**:123, 1904.

16. Bechterew, V.: Die Leitungsbahnen im Gehirn und Rückenmark, Leipzig, E. Besold, 1894.

17. Winkler, C.: Verlauf des Hackenbündels, Nederl. Vereinig. v. Psychiat. en Neurol. **11**:1 (Nov. 1) 1907; abstr., Neurol. Centralbl. **27**:94, 1908.

descending tract and a portion ascending to the posterior longitudinal bundle and to the nucleus of the fourth nerve.

Thomas [3] stated that the chief source of the so-called "faisceau en crochet" is the cortex of the vermis, as it shows extensive degeneration when this part alone is injured. It takes a position on the lateral surface of Gowers' bundle. After hemiextirpation of the cerebellum, the degeneration was bilateral. He followed the external arcuate fibers to the olives, to the nuclei of the raphe, to the lateral columns and to the reticular formation.

Fusé [4] described cerebellofugal fibers from the cortex to Deiters' nuclei. He described other fibers passing through the IAK in an arcuate course to reach the reticular formation at the level of the pons. They descend as far as the acoustic tubercle. Others pass to the posterior longitudinal bundle at all levels from the nucleus of the third nerve to the acoustic tubercle. He described another tract which lies lateral to the peduncles and partially corresponds to the "Hackenbündel." This arises from the cerebellar nuclei and ends in the reticular formation of the same and the opposite sides.

The dorsal bundle, however, shows as a distinctly separate tract. Its point of decussation is different—above the central nuclei as contrasted to the deeper position of the decussation of the ventral bundle. As it leaves the cerebellum it courses on the opposite side of the restiform body from that of the tract of Russel. The origin and in part termination of the two bundles are, however, much the same. As far as determined, the fibers of the dorsal bundle seem to lead to the midline nuclei, while the fibers of the ventral bundle pass to the vestibular system. According to Kappers,[18] these midline nuclei of the raphe and of the reticular formation are related to the posterior longitudinal bundle and subserve coordination of the cranial nerve mechanisms from the region of the oculomotor nucleus to the end of the bulb.

The literature reveals no recorded instance of injury to the cortex of the lingula. Most of the experimental work has dealt with lesions that involved the cerebellar nuclei. In the present experiments, lesions have been produced in more inaccessible parts, i. e., the lingula, and they have given clear evidence that many fibers arise in the cortex of this region and pass out through the neighborhood of the central nuclei. Moreover, a greater number of such fibers were found to arise in the more anterior parts of the vermis, i. e., the lingula, in contrast to those arising from the culmen and monticulus. It is of interest in this connection to note from the work of Ingvar [19] that the fibers of his direct

18. Kappers, C. U. Ariëns: Die vergleichende Anatomie des Nervensystems, Haarlem, de Erven F. Bohn, 1921.

19. Ingvar, Sven: Zur Phylo-und Ontogenese des Kleinhirns, Folia Neuro-biol. **11**:205, 1918.

vestibular tract ended for the most part in the more anterior and posterior parts of the vermis, i. e., the lingula, the uvula and the nodulus, and in the flocculus. Similarly, it has been shown by McNalty and Horsley,[20] and by Ingvar, that the ventral and dorsal spinocerebellar tracts end mainly in the lobulus centralis, culmen and pyramis of the vermis, while the medial part, as well as the lateral hemisphere, are free from such endings. Ingvar pointed out that these are the oldest parts of the paleocerebellum. The course of the ventral bundle is reciprocal to Ingvar's direct vestibulocerebellar tract, since it arises in the cortex of the lingula and inserts in the descending vestibular gray matter.

Brachium Conjunctivum.—Some fibers have been traced from the cortex of the anterior lobe of the vermis to the brachium conjunctivum, but none from the posterior lobe. A greater number of degenerated fibers appear when the globoid nuclei are injured. Still more degenerate following lesions of the dentate nuclei. These fibers ascend in the brachium conjunctivum and cross in its decussation, while those of cortical origin end in the red nucleus.

A cerebellotegmental tract is degenerated after lesions of the cortex of the anterior vermis. It follows the course of the fibers of the brachium conjunctivum to the point where they turn ventrally to form their decussation. Here the cerebellotegmental fibers pass dorsally and end about the large cells of the mesencephalic root of the fifth nerve.

The cortical origin of some of the fibers of the brachium conjunctivum has also been recently described by Sachs [21] and Löwy,[22] who reported degeneration in the brachium conjunctivum following lesions in the flocculi of rabbits.

SUMMARY OF SHORT CORTICOFUGAL FIBERS OF THE CEREBELLUM

The corticofugal fibers that arose from the vermis and passed to the central nuclei consisted of many fibers converging directly to these nuclei. Fibers from the inferior vermis travel largely to the roof nuclei and those from the superior vermis to the globoid nuclei. A separate compact tract passes from the anterior lobe to the lateral, dorsal and oral parts of the dentate nucleus. This is the only one of these corticonuclear connections that I shall discuss, as the others are already well known. Clarke and Horsley [2] described fibers that passed from the vermis to the forward end of the dentate nucleus. A number

20. McNalty, A. S., and Horsley, V.: On the Cervical Spinal Bulbar and Cerebellar Tracts, Brain **30**:237, 1909.

21. Sachs, Ernest, and Fincher, E. F.: Anatomical and Physiological Observations on Lesions in the Cerebellar Nuclei in Macacus Rhesus, Brain **50**:350, 1927.

22. Löwy, R.: Ueber die Faseranatomie und Physiologie der Formatio vermicularis cerebelli, Arb. a. d. Wien. neurol. Inst. **1**:21, 1916.

of workers have shown that this particular part of the dentate nucleus is phylogenetically the oldest. Gans [23] reviewed the literature on this subject and quoted from Weidenreich, Astwatzaturow and Vogt that in human fetuses this part of the nucleus was differentiated earliest. Van Valkenberg found that in a fetus of 42 cm. only this part was myelinated. Brouwer and Coenen,[24] in a case of congenital absence of the cerebellar hemispheres, found that only the dorsal part of the dentate nucleus was normal. Gans himself considered that the iron reaction of this part of the dentate nucleus indicated greater phylogenetic age. Kappers [18] claimed that in birds, before hemispheres are developed, it is possible to recognize a dentate nucleus in addition to the roof nucleus.

Association Fibers.—Association fibers are those that pass directly from one folium of the cerebellum to another. All parts of the paleocerebellum, the anterior and posterior lobes of the vermis, the lobulus simplex, the paramedial lobules and the flocculi are intimately connected by such fibers. There is no evidence of fibers from any of these parts leading to the hemispheres. Association fibers are most numerous in the parts nearest the primary sulcus, which are the phylogenetically younger parts. The nodulus and lingula are relatively poorly supplied with association fibers. Injury to the tuber and pyramis did not produce degeneration of fibers running to the anterior lobe, although injury to either anterior or posterior lobes did produce degenerated fibers running to the lobulus simplex. The flocculi are in association with both the anterior and the posterior lobes.

The area richest in association fibers is the medial lobe, which is the phylogenetically youngest part, while the anterior and posterior parts show few fibers of this type, but instead send long corticofugal fibers to the brain stem. Clarke and Horsley [2] showed that there are short association fibers between the folia in the hemispheres, and numerous long, anteroposterior connections in the medial part of the vermis. Brouwer and Coenen,[24] as a result of experimental work on rabbits and studies of the myelinization of the human fetus, recorded abundant association fibers between the flocculi and the vermis and between the different lamellae in the vermis. In an extensive experimental study, Saito [25] verified these observations.

COMPARATIVE ANATOMIC CONSIDERATIONS

The efferent tracts arising in the cortex of the lingula have considerable significance, since analogs have been recognized in the lower

23. Gans, A.: Beiträge zur Kenntnis des Aufbaues des Nucleus dentatus, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:750, 1924.

24. Brouwer, B., and Coenen, L.: Untersuchungen ueber das Kleinhirn, Psychiat. en neurol. bl. **3**:1, 1921.

25. Saito, Makato: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **24**:77, 1922-1923.

animals as the paleocerebellar system. Kappers [18] followed the development of the various efferent tracts phylogenetically as follows: Amphioxys has no cerebellum. This organ is first found in Petromyzon, in which it develops from the nuclei of the vestibular and lateral nerves. In the plagiostomes there is a vermis with auricles which receives fibers from the spinal cord and olives as well as from the vestibular system. There are two efferent tracts. The first is the brachium conjunctivum, which seems to arise from the subcerebellar cells, which are destined to migrate later into the cerebellum as the cerebellar nuclei. It crosses in the midbrain and ends in the red nucleus and the neighborhood of the oculomotor nuclei. The other efferent tract is the tractus cerebellomotorius, the fibers of which arise in the cortex of the auricles of the vermis and travel immediately beneath the ependyma of the ventricles and arch to the region of the posterior longitudinal bundle. This tract ends in the motor area of the bulb and midbrain. A closely related tract, called the "tractus cerebellovestibularis et cerebello-bulbares rectis" (Wallenberg), travels in the dorsolateral wall of the medulla. These are thought to be coordinating fibers between the cerebellum and the static equilibratory centers of the medulla. In the teleosts these two tracts have the same relations, except that an interauricular commissure is recognized in the velum. There is some evidence that in teleosts the brachium conjunctivum arises in part from the cortex. In reptiles, the tractus cerebellomotorius arises in the cortex of the vermis and possibly in part from the flocculi. The decussation in the velum is now traced to the "Hackenbündel" and from the crossed spinocerebellar tracts. The "Hackenbündel" descends in a dorsomedial part of the medulla near the ventricle. Its origin is said to be uncertain in reptiles. In birds, the cerebellar nuclei have completed their migration into the substance of the cerebellum and are said to give rise to the brachium conjunctivum and to the so-called cerebellobulbar and cerebellospinal tracts. These two tracts are said to arise from the mesial nucleus, and contain crossed and uncrossed fibers. The crossed fibers are said to be the homolog of the "Hackenbündel." The question has been raised whether the Purkinje cells do not, in birds, still give rise to fibers that will follow the older phylogenetic course. Since the present study has shown that homologs of the older cerebellar motor system persist in mammals, the question of their presence in birds might well repay reinvestigation.

The hemispheres and the vermis are first differentiated as the neocerebellum and paleocerebellum in mammals. The distribution of the fibers from the vestibular tract is limited to the lingula, nodulus and flocculi, and that of the fibers from the spinocerebellar tracts is limited to the lobulus centralis, culmen and pyramis. This distribution corre-

sponds with that found where these lobules constitute the whole of the cerebellar mechanism. Similarly, the older parts of the inferior olives are connected only with those lobules. New connections have arisen between the hemispheres of the cerebellum, the main olivary nuclei, the cerebrum and the pons. From the present study it is evident that an important number of the perforating fibers and fibers of the ventral and dorsal bundles in mammals take their origin from the cortex of the vermis and especially from the lingula, which is its oldest part, and that they still follow the old phylogenetic courses ending in the various vestibular nuclei, the old nuclei of the midline, and the gray matter of the reticular formation and that accompanying the posterior longitudinal bundle. Further, they arise from the same parts of the cerebellum in which the old spinal vestibular and olivary tracts end.

LOCALIZATION IN THE CEREBELLUM

The anatomic considerations lead to a view of localization of function in the cerebellum differing somewhat from that based on pathologic and physiologic grounds. The paleocerebellum, and especially the lingula or its extreme anterior part and the nodulus or its extreme posterior part and probably also the flocculus, maintains its old phylogenetic relations. It receives fibers from the inferior olives and vestibular nerves and their nuclei and fibers carrying proprioceptive stimuli from the spinal cord and cranial nerves. It sends out fibers to the vestibular system to the inferior olives, to the coordinating nuclei of the bulb and midbrain, such as the nuclei of the raphe, to the reticular substance and to the posterior longitudinal bundle. It subserves the phylogenetically old functions of coordinating static and equilibratory reflexes. The mesial part of the vermis is more recent in development, is very richly supplied with association fibers, and lesions here produce no symptoms in the rabbit (Ingvar[19]) or dog.[1] In all these respects this area resembles the so-called silent areas of the cerebrum. The hemispheres are connected mainly with the dentate nuclei, pons and cerebrum. It is important to bear in mind the phylogenetic relations of functions and to consider the anatomic relation of relays to the cerebellum in approaching the problem of localization, and a simple spatial projection of somatic areas such as has been suggested in the past will probably not suffice.

Some remnant of a simple anteroposterior pattern of spatial projection is indicated by the work of McNalty and Horsley,[20] who found that proprioceptive relays from the spinal cord subserving the neck and forelimbs are implanted chiefly in the anterior parts of the vermis, while those serving the hindlimbs enter mainly the posterior parts of the vermis. The supply to the intermediate portions is relatively small.

This conforms closely to the findings of the present work, in which lesions involving the intermediate portions (Ingvar's medial lobe) gave rise to extensive degeneration of short interlobular ("association") fibers but not to long fibers leaving the cerebellum entirely (corticofugal).

That there may be some definite localization of the connections between the cerebrum and the cerebellar hemispheres has been intimated by the physiologic and clinical work of van Rijnberk,[26] Holmes[27] and others, who have shown that lesions lying anteriorly in the hemispheres are apt to cause disturbances of the more oral parts of the body, and lesions lying posteriorly are apt to disturb the caudal parts of the body.

From his phylogenetic studies, Herrick[28] has developed a similar concept of cerebellar localization. He said, "Anatomical evidence suggests that any functional localization that may be present in the cerebellar cortex is an expression in the spatial distribution of efferent fibers from physiologically different lower centers."

SUMMARY

By the study of the Marchi degeneration in dogs with operative lesions in the cortex of the cerebellar vermis, it was possible to trace: (1) short cerebellofugal fibers from the cortex to the central nuclei; (2) long cerebellofugal fibers in four tracts to the various parts of the brain stem, and (3) intracerebellar association fibers to other parts of the paleocerebellum.

1. Short corticofugal fibers from the superior part of the vermis pass mainly to the roof nuclei. From the inferior part they pass mainly to the globoid nuclei, and there is also a definite well formed tract to the phylogenetically oldest part of the dentate nucleus.

2. Long corticofugal fibers from the vermis may be classified as follows:

(1) Fibers that enter the brachium conjunctivum to end in the opposite red nucleus or in the mesencephalic root of the fifth nerve.

(2) Perforating fibers that pass directly from the cortex to the vestibular nuclei, reticular formation, gray matter accompanying the posterior longitudinal bundle and descending tract of Deiters' nucleus.

(3) Fibers that arise from the most anterior part of the vermis (especially from the lingula) and take a position between the central

26. van Rijnberk, G.: Das Lokalizationsproblem in Kleinhirn, Ergebn. d. Physiol. **7**:653, 1908.

27. Holmes, G.: The Symptoms of Acute Cerebellar Injuries, Brain **40**:461, 1917.

28. Herrick, C. J.: Origin and Evolution of the Cerebellum, Arch. Neurol. & Psychiat. **11**:621 (June) 1924.

nuclei and ventricle where some of them decussate. Both crossed and uncrossed fibers enter the hooked tract of Russel and end in all of the vestibular nuclei and related gray matter, as do those of the second group. They follow especially the spinal vestibular nucleus and end in it at all levels. Some of these also follow the course of external arcuate fibers. To this group I have given the name of bundle of the ventral decussation.

(4) Fibers of similar origin that take a position dorsal to the central nuclei where they form a partial decussation. From here they pass to the lateral surface of the restiform body, where they ascend and descend and course as external arcuate fibers to reach the nuclei of the raphe at all levels and possibly also the olives. This is the bundle of the dorsal decussation.

3. Intracerebellar association fibers connect all parts of the paleocerebellum (vermis and flocculi), but these are least numerous in the oldest parts and most abundant in the phylogenetically younger, intermediate parts (i. e., the lobulus medius medianus of Ingvar).

ALTERNATING OCULOMOTOR PARALYSIS IN TRAUMATIC MIDDLE MENINGEAL HEMORRHAGE

E. S. GURDJIAN, M.D.
DETROIT

Alternating oculomotor paralysis, or Weber's syndrome, is characterized by palsy of the third nerve on one side with contralateral paralysis of the body. The lesion responsible for such a syndrome is located in the anterior aspects of the midbrain and involves the corticospinal path (pyramidal tract) and the fibers of the third nerve that traverse it. Alternating oculomotor paralysis may also occur in lesions of the middle fossa.

In this paper, I shall endeavor to show that acute traumatic middle meningeal hemorrhage may give rise to typical alternating oculomotor paralysis by virtue of the fact that the extension of the dissecting clot into the base of the middle fossa causes an oculomotor palsy, and its pressure against the convexity of the hemisphere is productive of contralateral paralysis of the body. From the surgical standpoint it is important to localize such a lesion accurately; otherwise, the treatment instituted may not alleviate the usually benign condition, i. e., extradural clot (unless there is associated severe brain damage).

REPORT OF CASES

CASE 1.—*Clinical History.*—R. H., aged 8, who entered the Receiving Hospital on April 3, 1931, on the night before admission, fell, striking his head on the pavement. He was dazed for an instant, but resumed play, walked home, had supper and went to bed. About 6 a. m. on the following day, he awakened with headache, nausea and vomiting. The vomiting was of the projectile type. In the course of three or four hours, he became progressively drowsy, and it was thought advisable to transfer him to a hospital. The patient was seen at 10 a. m., about sixteen hours after the initial trauma.

Examination.—The patient was a well developed boy, who was markedly drowsy, but could be aroused. There was a minor contusion in the right parietal region. There was ptosis of the right eyelid. The right pupil was dilated and was directed down and out; it reacted to light. There was slight edema of the right disk. The left pupil was constricted, regular and reacted to light; the left fundus also showed some edema. The patient favored the left half of the body. The tendon reflexes on the left were slightly diminished, and there was a positive Babinski sign. It was impossible to test the extra-ocular movements at this time.

Laboratory Studies.—The Wassermann reaction was negative; the urine was normal; the white blood cells numbered 20,600.

A diagnosis of an extradural middle meningeal hemorrhage was made.

Operation.—Two hours after entrance into the hospital, under ether anesthesia, a right osteoplastic flap was outlined, and an incision about 1 inch (2.5 cm.) long was first made in the line of the flap. On boring a hole through the bone the meningeal clot was encountered. The osteoplastic flap was turned down, and an area, 5 inches (12.7 cm.) square, of the dura was exposed. There was a fracture of the parietal bone, 4 inches (10.1 cm.) long, extending into the base of the middle fossa. A large clot, 4 by 1½ inches, was removed from the epidural space. All bleeding points were stopped. It was thought that the artery was injured at a level about 1 inch beyond its exit from the foramen spinosum. This was in close proximity to the point where the artery divided into its two terminal branches. The artery was ligated. A subtemporal decompression was performed, and the skin was closed in layers. A drain of gauze was placed in the epidural space and brought to the surface through one of the drill holes, being removed on the following day.

Fig. 1.—A portion of the clot removed in case 1. It weighed 65 Gm. Note that its removal *en masse* would not have been easy through the subtemporal approach.

Course.—Progressive amelioration of the condition ensued. The temperature remained around 101 F. for four days. The state of consciousness was good on the day following the operation. The ptosis disappeared practically completely within a week after the operation. Still later there was no evidence of ptosis on the right, and the extra-ocular movements were normal.

Comment.—In this case there was a genuine Weber's syndrome, with oculomotor paralysis on the side of the lesion and contralateral weakness of the body. Ether anesthesia was used in order to increase the intracranial pressure sufficiently to cause herniation of the brain to a normal position.

The convalescence was short. The patient was up and about within ten days. I think that the flap operation is responsible for such a quick response. Drainage is definitely indicated, for it enables one to remove

any postoperative hemorrhage on the day following the operation. Such hemorrhage practically always must occur, for the space between the dura and bone is large and may facilitate bleeding not only from the meninges, but from the bone flap. A general anesthetic obliterates some of this space by increasing the intracranial pressure.

CASE 2.—*Clinical History.*—H. S., aged 41, entered the Receiving Hospital on April 12, 1931, with a history of having been struck on the head a few hours before. On entrance, she was semiconscious and was aroused only with insistence.

Examination.—There was swelling over the right parietal region with many abrasions and lacerations, which had been treated in the emergency department. No evident depressions were felt. The right eyelid showed ptosis, particularly when the patient was aroused, as she opened the left eye, while the right eye remained closed. The right pupil was dilated and fixed to light. It was round and regular. It pointed laterally and downward. The right disk showed some engorgement of the blood vessels, but no definite edema was made out. The left pupil was round and constricted, but reacted to light. The patient was too ill for testing the extra-ocular movements. There was cortical facial weakness on the left. The tongue protruded in the midline. There were no injuries to the thorax and abdomen. The blood pressure was 125 systolic and 70 diastolic. The tendon reflexes were present and normal on the right; they were somewhat diminished on the left, and the Babinski phenomenon was present. On observing the patient for several minutes, it was evident that she used the right half of the body much better than the left.

A diagnosis of extradural middle meningeal hemorrhage was made.

Laboratory Examinations.—The Wassermann reaction was negative; the white blood cells numbered 19,500; the urine was normal. Repeated roentgen examinations did not reveal a fracture.

Operation.—On April 14, 1931, a right subtemporal decompression was performed. In view of the presence of multiple lacerations in the right parietal region, the usual osteoplastic craniotomy was not resorted to, because of the possibility of infection. Through a large opening in the right temporal region, the greater portion of the blood clot was removed. All bleeding points were stopped, and the incision was closed in layers. A small gauze drain was left in the subdural space, to be removed on the following day. The operation was performed with the patient under local anesthesia. When the clot was removed, there was a space about 1 inch in thickness between the dura and the bone. The brain and dura were pushed upward and medially by the growing blood clot. It seemed that the middle meningeal artery was lacerated just as it came out of the foramen spinosum. No fracture was made out at the site of the operation.

Course.—Gradual amelioration of the patient's condition ensued. With her increasing consciousness and interest in the environment, the ptosis of the right eye became more evident. Careful examination after the patient was able to cooperate showed that there was a complete paralysis of the third nerve and a partial paralysis of the fourth nerve on the right side. In about three weeks, the patient was able to elevate the upper lid voluntarily, but in the resting state the lid came down again. About four months later there were no evidences of extra-ocular palsies, and the patient apparently was well.

Comment.—The neurologic symptoms in this patient were sufficient to justify a diagnosis of Weber's syndrome. The oculomotor paralysis

Fig. 2.—A photograph of the patient in case 2, two weeks after the operation. The right eye shows evidences of paralysis of the third nerve. The ptosis was more marked before the operation. No appreciable proptosis was evidenced in this case. Note that the left lower part of the face is smoothed out in a manner characteristic of weakness of the lower part of the left side of the face. This patient showed also some weakness of the fourth nerve by virtue of the fact that the eyeball was turned out and not down.

Fig. 3.—The patient in case 2, four months after the operation. The ptosis had disappeared practically completely, and the patient was able to rotate the right eyeball in all directions.

The photograph on the right shows the right eye turned in and up, showing evidence of activity of the inferior oblique and internal rectus muscle.

on the side of the lesion and contralateral paresis warranted such a conclusion. The usual osteoplastic flap could not be made in this case because of many lacerations in the right parietal region. Therefore a portion of the clot was removed through a large subtemporal opening. The convalescence was long. The patient was in the hospital for five weeks. This undoubtedly was due to the fact that the flap operation was not performed. It is possible for a middle meningeal hemorrhage to occur without an accompanying fracture of the skull. However, I am cognizant of the fact that the fracture may be altogether too insignificant and therefore not be shown by the x-rays. In one case, not included in this group, there was a crack of the skull, 0.5 cm. long, in the vicinity of the foramen spinosum.

Case 3.—*Clinical History.*—D. B., aged 28, entered the Receiving Hospital on July 20, 1931, in an unconscious state and with stertorous breathing. His wife stated that about fifty-six hours before admission he was driven home after having been hit on the head with a timber. He was conscious when he entered the house, and he went to sleep that night, waking early the next morning with headache and nausea. He was up all day on the day after the accident, but complained of terrific headache, and it was also noticed that the right eyelid drooped slightly. On the following morning, he had a generalized convulsion which eventuated in unconsciousness. His condition became progressively worse. He was brought to the Receiving Hospital at about 2 a. m. When seen the next morning, the patient was in deep coma, but he reacted to painful stimuli.

Examination.—The temperature was 100 F.; the pulse rate was 60, and the respiratory rate, 25. There was a slight abrasion in the right parietal region. The right eyelid was swollen and slightly ecchymotic. On opening the right eye, it was noted that the pupil was dilated and the eyeball turned out and down. The fundus showed definite choking to the extent of 1½ diopters. The left pupil was constricted, but reacted to light. The eyeball was turned slightly inward. The fundus showed slight swelling, but no measurable choking. On painful stimulation, the patient showed weakness of the left lower part of the face, but this was not a dependable observation. The chest was filled with coarse râles, and a beginning dependent pneumonia was diagnosed. The tendon reflexes in the upper extremities were equal. In the lower extremities there was a bilateral abortive patellar clonus, which was more marked on the left side. There was also a bilateral Babinski sign, also more marked on the left. There was no ankle clonus. Roentgen examination showed a fracture line in the right parietal region, extending into the middle fossa. The white blood cells numbered 17,400, with 81 per cent polymorphonuclears.

On the basis of the dilated right pupil and evidences of paralysis of the third nerve and fracture of the right parietal region, a diagnosis of middle meningeal hemorrhage was made.

Operation.—An osteoplastic flap was outlined in the right parietal region, and an incision was made in a line parallel with this outline. A hole was drilled through the bone, and a meningeal clot was encountered. After corroboration of the diagnosis, the osteoplastic flap was turned down and an area of 5 by 5 inches of the dura was exposed. The entire surface was covered with blood clots, with evidences of beginning organization. The clot had dissected its way between the dura

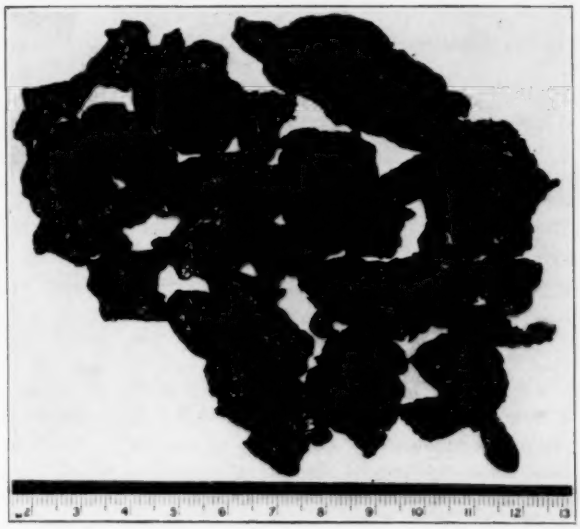

Fig. 4.—A portion of the clot removed in case 3. It weighed about 73 Gm. The entire clot could not be removed *en masse* because of beginning organization.

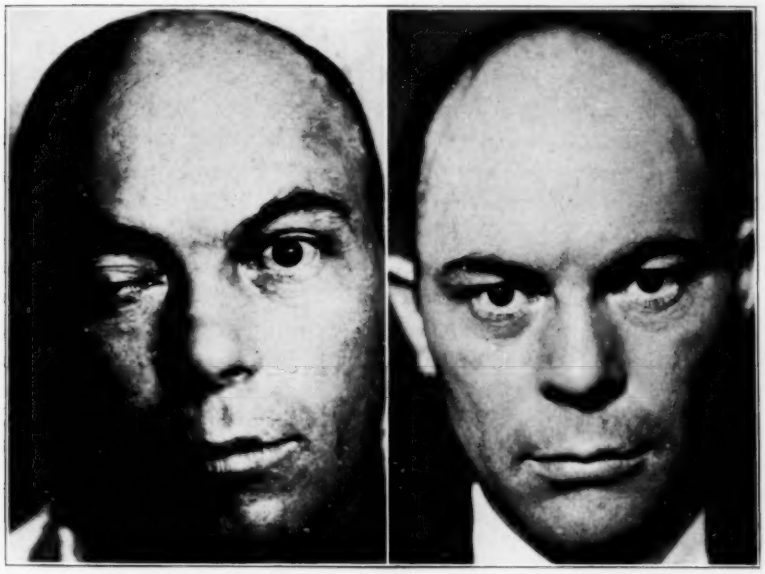

Fig. 5.—The photograph on the left shows the patient in case 3, ten days after the operation. There was some improvement in the extra-ocular condition, although the ptosis and other evidences of paralysis of the third nerve were still marked. The photograph on the right shows the same patient, three months after the operation. Complete restoration of extra-ocular movements is noted. In all three cases reported, the dilatation of the pupil disappeared in a short time and reaction to light was obtained. Note the anterior limb of the horseshoe incision used for the craniotomy.

and the bone into the base of the middle fossa. It also had extended up and medially, and actually had crossed the midline with the formation of a globular mass, about 1 inch (2.5 cm.) in thickness, at the midline. On removal of the blood clot, it was noticed that the artery was most probably injured at the point where it divides into anterior and posterior branches. After removal of the clot, all bleeding points were stopped and a gauze drain was placed in the epidural space, coming through one of the drill openings. The incision was closed in layers.

Course.—Progressive amelioration followed in the condition of the patient. About 1 a. m. on the night of the operation, the temperature rose to 103 F., with an increase in the pulse rate to 145. The gauze drain was partially pulled out, and this allowed the discharge of tarry, black blood. Following this procedure, the condition was much better, so that on the following morning the temperature went down to 99 F., and the patient uttered a few words. The subsequent course was uneventful. With oncoming consciousness and cooperation of the patient, it was evident that he had a complete paralysis of the right third nerve, with ptosis of the lid, dilated pupil and out and down rotation of the eyeball. The ptosis became much better before the patient left the hospital on July 30. Since then, he has had no headaches. When seen on Aug. 24, 1931, the ptosis had practically completely disappeared, and there was return of extra-ocular movements indicating a return of function of the third nerve. Still later extra-ocular movements were normal (fig. 5).

Comment.—Case 3 is an example of middle meningeal hemorrhage with paralysis of the third nerve on the same side and evidences of irritation of the pyramidal tracts on both sides. The more marked involvement of the lower extremities and the pathologic conditions observed at operation remind one of the longitudinal sinus syndrome discussed by Holmes and Sargent [1] (1915). The pathologic observations show the reason for the evidences of irritation of the pyramidal tract in the lower extremities on both sides. The dissecting clot, undoubtedly aided by bleeding from the parietal emissary veins and bone vessels, caused the formation of a mass large enough to press against the lower extremity centers of the cortex on both sides. The paralysis of the third nerve encountered in this case was an expression of the dissecting clot finding its way between the bone and the dura at the base of the middle fossa and pressing against the contents of the superior orbital fissure.

COMMENT

Traumatic middle meningeal hemorrhage is a well known clinical entity. The contributions of Krönlein [2] (1885), Jacobson [3] (1886), von

1. Holmes, G., and Sargent, P.: Injuries to the Superior Longitudinal Sinus, Brit. M. J. **1**:493, 1915.

2. Krönlein: Ueber die Trepanation bei Blutungen aus der Arteria meningea und geschlossener Schadelkapsel, Ztschr. f. Chir. **23**:43, 1885.

3. Jacobson, W. H. A.: On Middle Meningeal Hemorrhage, Guy's Hosp. Rep. **28**:147, 1886.

Bergmann[4] (1904), Cushing[5] (1912) and others are evidently the result of exhaustive studies and accurate observations on the subject. Plummer[6] (1896) and others discussed the anatomy and variations of the middle meningeal artery. The chapter on middle meningeal hemorrhage in von Bergmann's "System of Surgery" is comprehensive, and the present day student will find it just as enlightening as, if not superior to, recent discussions. Particularly in autopsy material in cases of death from skull fracture, the incidence of middle meningeal hemorrhage is high. Moody[7] (1920) and Lecount and Apfelbach[8] (1920) reported 100 cases of epidural bleeding, most of which were of meningeal origin. According to Moody, the condition was not suspected clinically in 63. In 37 it was recognized, and the patients were operated on (subtemporal decompression). Of these, 26 died, 18 within the first twenty-four hours after the operation. Clinically, all but 2 showed signs indicative of involvement of the brain, such as hemiplegia, unilateral rigidity, convulsions, etc. Vance[9] (1927) noted 61 cases of epidural bleeding in 512 autopsies. In 11 of his series, an operation was performed, with 8 deaths and 3 recoveries. The pupil was dilated on the side of the lesion in all but 1 case. According to Vance and Moody, the most usual points of laceration of the artery are: (1) at the emergence of the middle meningeal from the foramen spinosum, and (2) where the artery divides into its anterior and posterior branches.

The diagnosis of middle meningeal hemorrhage is simple in the typical case. The usual lucid period preceding the progressive drowsiness is pathognomonic, although cortical lacerations and localized pressure changes in the subarachnoid space may simulate this syndrome (Peet,[10] 1927). Unlike cases with laceration of the brain, extreme increased intracranial pressure and traumatic encephalitis, the middle meningeal clot is usually associated with a temperature slightly above normal (from 99 to 102 F.). In the beginning, the pulse rate is not rapid; in fact, it ranges from 50 to 70 per minute. The patients are

4. von Bergmann, E.: System of Practical Surgery, Philadelphia, Lea Brothers and Coe, 1904, vol. 1, p. 206.

5. Cushing, H.: in Keen: Surgery, Philadelphia, W. B. Saunders Company, 1912.

6. Plummer, S. C.: Surgical Anatomy of the Middle Meningeal Artery, Ann. Surg. **23**:540, 1896.

7. Moody, W. B.: Traumatic Fractures of the Cranial Bones, J. A. M. A. **74**:511 (Feb. 21) 1920.

8. Lecount, E. R., and Apfelbach, C. W.: Pathologic Anatomy of Traumatic Fractures of Cranial Bones, J. A. M. A. **74**:501 (Feb. 21) 1920.

9. Vance, B. M.: Fractures of the Skull, Arch. Surg. **14**:1023 (May) 1927.

10. Peet, M. M.: Symptoms, Diagnosis and Treatment of Acute Cranial and Intracranial Injuries, New York State J. Med. **28**:555 (May 15) 1928.

drowsy but can be roused; the general impression is that they are in a good condition when first seen.

One of the most constant symptoms in middle meningeal hemorrhage is the dilated pupil on the side of the lesion. The significance of the dilated pupil in these cases seems to be poorly appreciated; its causation is not enlarged on in most of the recent papers I have consulted. I think that it is brought about by pressure of the enlarging blood clot against the extra-ocular nerves. When one notes the direction in which the clot dissects its way between the dura and the bone, it becomes easy to understand the mechanism of production of palsy of the third nerve in these cases. With the clot originating in the vicinity of the foramen spinosum, its extension medially and forward in the middle fossa causes enough pressure against the contents of the superior orbital fissure to produce paralysis in the oculomotor distribution. Its extension posteriorly toward the posterior fossa is checked by the presence of the tentorium cerebelli. The cavernous sinus is fairly well protected by the gasserian ganglion and its branches. The fourth nerve was involved in one of my cases, as shown by the fact that the eye at times was merely pulled outward by the overactivity of the lateral rectus, while the simultaneous downward movement of the eyeball caused by the superior oblique muscle was missing. The trochlear nerve in its course through the cavernous sinus is in close proximity to the third nerve. It is surprising that all cases do not show trochlear derangement. This may possibly be explained on the basis that the trochlear nerve is not as sensitive as the third nerve, and furthermore the latter, owing to its larger size, may protect the trochlear against the invading clot. The sixth nerve has escaped damage in my cases, and this may be due to the fact that in its course through the superior orbital fissure it is protected by the reflected edge of the dura because of its lateral position. Posterior to the superior orbital fissure it is deeply placed in the cavernous sinus and is in close proximity to the bone. In this position it is difficult to dislodge without doing much damage to the cavernous sinus region. Von Bergmann [4] stated that abducens paralysis may occur in middle meningeal hemorrhage, and this is undoubtedly possible.

It should be emphasized that, although the clot is initiated by bleeding from the middle meningeal artery, the smaller vessels and capillaries of the dura and bone aid in its formation and further enlargement as the process of dissection of the dura off the bone proceeds. The base of the middle fossa is very vascular, and bleeding from blood channels in this region is undoubtedly responsible for the production of the extra-ocular palsies. In case 3, the large globular mass crossing the midline at the vertex was probably mostly derived from bone and dural vessels other than middle meningeal branches.

I think, therefore, that the dilated pupil in middle meningeal hemorrhage is most frequently an expression of paralysis of the third nerve due to pressure of the clot against the nerve in the middle fossa. Close scrutiny will also reveal that not only is the pupil dilated but the eyeball is turned out (owing to abducens overactivity) and down (owing to trochlear function), and that there is an associated ptosis that escapes notice because of the usual seriousness of the patient's condition before operation. In two of the cases reported here, the ptosis was noticed before operation, in one case by the wife and in the other by me. If there is an associated contralateral paralysis of the body, which may be expected from the extension of the clot over the convexity of the hemisphere and by virtue of the fact that the hemisphere is pushed up and medially in the motor area, the similarity of such a symptom complex with that described by Weber becomes apparent. Weber's syndrome, or alternating oculomotor paralysis, is of midbrain significance, and therefore, neurosurgically speaking, a posterior fossa lesion. The alternating paralysis of middle meningeal hemorrhage is not due to derangement of the midbrain, and it is a middle fossa lesion. From the standpoint of operative treatment, this is a significant fact.

Moody [7] (1920) pointed out that the white cell count in cases of middle meningeal hemorrhage is very high, and, therefore, might be used as a criterion in diagnosis. The cases described showed a white count ranging from 17,000 to 21,000. It must be remembered, however, that such high counts are not infrequent in cases of fracture of the skull with injury of the brain and no middle meningeal hemorrhage, either at operation or at autopsy.

A few words are advisable concerning the operative approach in these cases. The older investigators busied themselves about the diagnosis of the exact location of the clot and trephined over that area. Thus, they would operate more anteriorly for clots arising from the middle meningeal artery before it divides, more posteriorly for hemorrhages originating from rupture of the posterior branch of the artery, etc. Krause [11] (according to von Bergmann) suggested that his low flap operation used in gasserectomy should be employed for removal of the clot. These various methods have been supplanted, in this country at least, by the well known subtemporal decompression of Cushing. Thus, in the cases described by Moody and Vance, in which an operation was performed, a subtemporal decompression was done. In a certain number of cases such an approach may be sufficient, but it must be remembered that the clot may be far beyond one's reach through an opening in the temporal fossa. For this reason I prefer an osteoplastic flap. This exposes the entire clot, and its removal

11. Krause, quoted by von Bergmann (footnote 4).

becomes simple. It will also allow the operator to investigate easily the base of the middle fossa. The operation of an osteoplastic flap takes no longer than subtemporal decompression. Furthermore, the period of convalescence after the flap operation is much shorter, as is to be expected from the fact that with such a procedure the entire clot is removed. Through a subtemporal approach a very small portion of the clot is removed, and if the patient survives the remainder has to be absorbed, thus lengthening the period of recovery. After outlining the flap, I prefer to make a small incision in line with the outline and to drill a hole through the bone to note whether or not the diagnosis is correct. Following the confirmation of the diagnosis, the flap operation is completed. I believe in leaving a small drain in the epidural space, bringing it out through one of the drill openings in the bone. This is removed on the following day, and I think that it provides a good means of removing secondary operative hemorrhage in the epidural space. Of course, if it is found expedient to open the dura, the latter should be well repaired. I prefer a general anesthetic with ether in such cases, because it affords some increase in intracranial pressure, which pushes the dura back to a normal position. Under local anesthesia, as in one of my cases, after the removal of the clot a big gap between the bone and the dura remains empty, and I think that such a condition might facilitate hemorrhage.

CONCLUSIONS

1. Middle meningeal hemorrhage may cause alternating oculomotor paralysis.

2. The dilated pupil in meningeal hemorrhage is usually the result of palsy of the third nerve, and one should also look for possible extraocular paralyses and ptosis.

3. When the diagnosis of middle meningeal hemorrhage is made, an osteoplastic flap should be the operation of choice. It is always wise to drill a little hole or two in the bone to confirm the diagnosis. These openings should be made in close proximity to the ear, so that a possible clot that has not extended further than the base of the middle fossa may not be missed.

TACTUAL PERCEPTION IN ALCOHOLISM

STUDY OF THE INFLUENCE OF ALCOHOLIC AND OF OTHER PSYCHOTIC STATES ON TACTUAL AFTER-EFFECTS

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When the perception of an uncomplicated tactual or visual sensation is retained in consciousness for a long time, certain definite changes in this perception are observed. Similarly, when an imagined sensation is retained in the same manner, changes also occur. By studying the development and fate of these changes in the visual and tactual after-effects or imaginations, insight can be obtained into the structure and determinants of perception.

In the sphere of visual phenomena a careful investigation was made by Kanner and Schilder [1] of the movements in the representation of optic images and the representation of optic images imagined to be in movement. They have described the results for the images of both quiet, inanimate objects and those in natural movement. They found many tendencies toward movement chiefly of a waving character, scintillation of images, multiplication, changes in size and shape, disintegration and fragmentation of lines, fading and diffusion of light occurring as an inherent tendency in the process of representation of optic images. The results were roughly similar for inanimate or animate objects in all subjects, but showed individual variations in single subjects. Apparently it made no difference whether the representation was of a memory image or of an after-effect.

These results provide a striking situation, for they show how closely the characteristics of optic perception in imagination correspond with those in the after-effects of visual images and in eidetic images. In the studies of eidetic phenomena initiated by Jaensch (best described as phenomena midway between memory images and after-effects), certain definite movement tendencies in these images were observed in some persons. On the basis of this, two psychologic reaction types were described—the tetanoid (T type), in which eidetic images were in general static, and the basedowoid (B type), in which the images were moving.

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1. Kanner, L., and Schilder, P.: J. Nerv. & Ment. Dis. **72**:489, 1930.

Schilder and I [2] showed that when normal subjects retained tactual after-effects of simple geometric patterns, as a spot, a cross, a triangle, etc., or when they imagined receiving sensations of this character, changes took place that can be summarized in a general way as follows: After-effects and imagined after-effects had a tendency to move rhythmically, to diffuse, to be partly effaced or broken up, or to form units (Gestalten) and to undergo transformations of size and shape. All these tendencies, especially those of movement, showed an increase in activity when the subjects were experimentally put under the influence of vestibular irritation by turning in the Bárány chair. Vestibular irritation, then, brings into the foreground of perception those products of tactual disintegration that usually are given little attention in ordinary tactual perception. It was found that although tactual images followed optic images at the outset, the former developed along widely divergent lines in time from the latter, and this was especially true under the influence of vestibular stimulation. It was observed that products of disintegration in the tactual after-effects in the early experiments were strikingly similar to the tactile images developed after vestibular irritation.

It was pointed out that the elements of sensory disintegration found in the patients with peripheral nerve lesions or cerebral intoxications (mescal poisoning) were closely similar to the experimental results obtained. The presence of these movement tendencies, etc., in optic, eidetic and tactual images seems to indicate that these elements may be inherent in the process of perception per se. A hypothesis was suggested that the process of integration of perception utilizes the same elements and tendencies as occur in the disintegration of images, and that the particular elaboration depends on the psychic disposition of the individual person. It has been further maintained by Schilder that pathologic perception in lesions of the central nervous system represents but isolated phases of what goes to form the developed perception in normal persons. From this point of view it seemed indicated to study the fate of tactual after-effects in psychotic patients with tactile hallucinations, as in delirium tremens and other types of psychoses.

MATERIAL

The material used in this study was from the active psychopathic and alcoholic service at Bellevue Hospital. The patients included sixteen with alcoholic psychoses (delirium tremens, acute hallucinosis, confused delirious state, "wet brain"), ten with schizophrenia (paranoid and hebephrenic types), one with manic-depressive psychosis in a manic phase and one with senile psychosis (paranoid type). The

2. Bromberg, W., and Schilder, P.: On Tactual Imagination and Tactual After-Effects, J. Nerv. & Ment. Dis. **76**:1 (July) 1932.

patients showing tactile hallucinations were in the group of alcoholic and schizophrenic persons.

Many difficulties were present in working with these patients. Some were uncooperative and resistive or preoccupied with delirious or hallucinatory mental content. The type of patient was not such as is given to psychologic introspection. For these reasons no attempt was made to test tactual imaginations.

The procedure followed was, with some modifications, that developed by Schilder and myself. The patients were asked to close their eyes and report everything they felt. They were then touched bluntly once on the hand or forehead. Later, two spots, successively or simultaneously, were made. Then a line, triangle, cross or circle was drawn. The patient was urged to hold the tactual image as long as possible and asked to try to exclude optic impressions.

In the description of the observations, for the sake of brevity, only portions of some of the protocols will be presented to illustrate the points discussed.

EXPERIMENTAL RESULTS IN CASES OF ALCOHOLISM

Although in this group there were cases of delirium tremens, alcoholic hallucinosis, alcoholic wet brain with polio-encephalitic signs and acute intoxications in persons with chronic alcoholism without psychoses, there was no qualitative difference in the tactual responses obtained in these different pictures. The long duration of the after-effects was surprising in this group compared to the average of from two to three minutes in normal subjects. In the delirious patients, the impressions often lasted as long as five minutes; in one of the patients with nonpsychotic chronic alcoholism they lasted fourteen minutes. It was interesting from the standpoint of theoretical considerations, that as the condition cleared up the time of the after-effects correspondingly decreased. In addition to the reduction in duration and intensity, the elaboration of the tactual sensations was inversely affected by improvement in the psychiatric and somatotoxic picture of the alcoholic psychoses (case 3, experiments 1, 2 and 3). It may be that the prolongation of the after-effect in some cases (case 2, experiment 1) was involved with the memory image of the sensation, for in the study with normal subjects one was found whose after-effect was replaced by a memory image. Similarly, another patient (case 3, experiment 2) felt that he was prolonging the after-effect because of his imagination (memory image) of the object. But in most of the alcoholic patients the long duration of the after-effect is probably related to the direct effect of the alcoholic cerebral intoxication.

One of the outstanding observations was the feeling of movement, especially of rapid movement, in the after-effects. At times the movement had a rotary character (case 3, experiments 2 and 4). Often, rapid diminution in the size of the objects seen and felt was associated with the movement tendency. Sometimes the after-effects took their form from the content of the delirium, as in the patient in case 4, experi-

ment 4, in whom the movements and multiplicity of tactual sensations were felt as insects in vivid tactual hallucinations. The sensation actually drawn on the skin was soon broken up into many fragments, or it was felt as a single impression that multiplied later in the form of a line. The patients often felt numerous drops or particles on the skin and thought that they were connected somehow with water. It was interesting to observe that in case 4 there was a decrease in the rapidity of the after-effects with the improvement in the mental picture, as seen at successive examinations (experiment 1 and 6).

To evaluate and interpret these observations, one must consider the relation between the optic and the tactile sphere and vestibular irritation. This is the more necessary since Bárány and others have shown that alcohol influences the cerebellar and vestibular systems. Experimentally, in this clinic we have seen the direct influence on tactual after-effect of vestibular irritation by turning the subject in the Bárány chair. Clinically, in various conditions, confirmation of this phenomenon can be seen. Hoff and Schilder [3] described a syndrome of parieto-occipital lesion associated with multiple hallucinations when the patient turned on his longitudinal axis. In a case of labyrinthine disease, Eisinger and Schilder,[4] analyzing the patient's dreams, found in them similarities to delirium tremens. Recently, Hoff [5] reported the case of a patient who, following head trauma, developed epileptic seizures preceded by an aura in which objects appear frayed and flattened. The patient developed left-sided signs with left hemianopia; the aura then consisted of waves seen in the hemianopic field, followed by nystagmus to the right. Operation revealed a cyst directly over the right angular gyrus. The author intrepreted this to mean that the angular gyrus protects the visual area from the penetration of vestibular impulses to it; a lesion there, however, allows the vestibular impulses to reach the visual area and give rise to distorted optic images. A case in this series shows similarly the distortion of optic images under the influence of vestibular irritation.

CASE 1.—Do, a woman, aged 35, suffering from chronic alcoholism, was admitted to the hospital after an alcoholic spree for a week. She was agitated and tearful, but talked freely. The breath was markedly alcoholic; the gait was unsteady; the patient was toxic and had a rapid pulse. She had a vivid hallucinatory psychosis on an alcoholic basis, which she soon cleared up. The patient said on admission, "I saw an armless, legless woman floating behind me on tissue paper wings. I saw the rottenest thing I ever saw in my life—my friend was flying about in a small airplane but she was bodyless . . . there was another girl in the plane with half a body. . . . A policeman sat on her face and made it flat as a pancake. I swear on the Bible that I saw it. I'll bet she comes into

3. Hoff, H.; Schilder, P., and Gerstmann, J.: Opticomotor Syndrome of Rotation Around Longitudinal Axis of Body, Arch. f. Psychiat. **76**:766, 1926.

4. Eisinger, K., and Schilder, P.: Monatschr. f. Psychiat. **73**:314, 1929.

5. Hoff, H.: Ztschr. f. d. ges. Psychiat. u. Neurol. **121**:751, 1929.

the hospital with a reconstructed face. . . . Everything I see is pretty—the girl with the half body was pretty. . . . I felt sorry to see the girl without a body."

Temperature sensations were felt in the after-effects by practically all of the patients. Sensations of heat, the commonest type, were more intense than in normal subjects; they were painful at times and in case 4, experiment 1, and in case 5, experiment 2, the temperature sensation was an inflicted pain fitting into the course of action of the delirium. When two simultaneous touches were made, the tactual images tended to come together as in normal subjects. In case 3, experiment 4, the two sensations were felt immediately as one, showing the vestibular influence expressed in terms of increased rapidity of movement.

Comment.—According to experiences with normal persons, all these movement tendencies can be considered as products of disintegration of tactual sensation. It is known, however, that such disintegration is especially present when the vestibular system is affected. It seems justified, therefore, to suppose that the particular character of the perception in persons suffering from alcoholism is due partly to vestibular influences. But there is no proof that this is the only cause, because it is known that occipital lesions make for optic disintegration and parietal lesions increase tactual disintegration. In view of this, it is possible that tactile disintegration is due partly to cortical changes in perception. Bleuler considered tactile hallucinations in connection with paresthesias and was inclined to place them on a neuritic basis. I do not know how the disintegration described here can occur on a neuritic basis. But even if Bleuler's hypotheses were verified, one could still state that the disintegration occurring in the after-effects would be merely emphasized by the pathologic nerve changes.

Case 2.—Eg, a man, aged 38, suffering from alcoholism for many years, was admitted after a prolonged drinking bout. He was ataxic, flushed, noisy, boisterous and irritable; there were no trends or hallucinations.

Experiment 1.—Triangle drawn on the hand: "It's kind of centering. In the triangle now I got that dot from the previous experiment. The triangle is about the same. I know the points are there. The triangle came back at the same time the cross came back. . . . The spot went away when I concentrated on the triangle. When I concentrated on the points, I felt the points." (The sensation lasted about five minutes.) "I can prolong it by 'imagination.'"

Case 3.—Le, a business man, aged 61, addicted to alcoholism for forty years, was admitted after a prolonged debauch. He was excited, irritable and boisterous. He had no hallucinations or trends. He was in a moderate state of shock with cyanosis, rapid pulse and midbrain involvement. Experiments 1 and 2 were performed on the day of admission, Feb. 25, 1931.

Experiment 1.—Cross drawn on the dorsum of the right hand: "That's a cross. It is bordering on an X. Feels cool now. I know where the crossing point is but there is no feeling. Now it is going up the arm. It's up to the elbow. Now

it's down on the two fingers (pointing to fifth and fourth fingers). A cross with a long vertical line. . . . There is a slight burn at the wrist in the shape of a cross. . . . Felt the whole cross but some parts stand out which change. . . . Now it's spreading at the upper end of the cross to encircle the forearm in a broad band." (Duration more than eight minutes.)

Experiment 2.—Triangle drawn on the right hand: "That's a triangle with its base toward the knuckles. It's warm. . . . The lines are spreading out broader, but less intense. There is a spot close to the knuckle of the middle finger away from the triangle. The triangle is still there. In the center of the triangle is a slight ache. The ache is spreading toward the wrist. . . . It feels as if the whole triangle were turning so apex were going toward the knuckle of the ring finger. I feel the angles better than the sides, but dull. . . . Triangle is smaller. At first each side was 1½ inches, now they are 1¼ inches. I don't know whether it is memory or imagination, but there is still some sensation left." (Duration fourteen minutes.)

On February 26, the patient's condition was better. He was quieter and more cooperative. The physical condition was better.

Experiment 3.—Triangle drawn on the right hand: "An inverted triangle. The base is toward the knuckles (corner). Kind of a coolness. I can feel the triangle; can feel sharp angles. It is just like they were fading out. In general, it's getting weaker. I can feel the two angles at the base; feel both ends of the top line. It seems to be contracting somewhat slightly. Still seems to be pricking. The triangle has changed, more of a circular form." (Duration four and one-half minutes.)

Experiment 4.—Two dots simultaneously with the fingertips 1½ inches apart: "One impression with a division between it. The division is about $\frac{1}{10}$ of an inch. It's kind of a long oval. It seems to be warming up a little. It seems as though it were kind of twisting and the outside one going down faster than the other, like rotating. The rotation is increased. . . ."

Case 4.—Ho, a laborer, aged 45, had been drinking for several days. A delirium followed a fracture of the right leg. The patient had numerous tactile and visual hallucinations, with a trend reaction. On admission on Jan. 21, 1930, he was elated and talkative. He stated: "The Jews threw lice at me. It takes four months to catch a mother louse and I've the mother louse. They threw them right in my beard. They are white ones and black hairs. A mother louse controls them. They will come out pretty soon. Watch at 6 o'clock."

Experiment 1 (Jan. 22, 1931, 9:30 a. m.).—Cross drawn on the right hand: "That's a cross. Somebody is punching crosses. I don't know how many. Still punching crosses, little crosses. They are coming right out of the little finger. The finger is frozen; something is holding it there. It's a cut or something, from the little joint to the upper joint of the little finger. It's coming out in one stream. It's coming out through the center of the cross in one stream." (Duration five minutes.)

Experiment 2.—Circle drawn: "It's going around in a circle. Somebody is punching my wrist. There is still a mark there. Somebody is pressing on it. It's going deeper and deeper and deeper. No sensation at all. I feel something coming out—water. I don't feel anything." (Duration, three minutes.)

Experiment 3.—Touch made on dorsum of hand: "Punching something on the vein. Now you are punching something on the same vein. It must be coming up my arm—something stiff. My arm is getting longer; it's going forward. The finger is getting longer. The little finger is the first one, then the middle, then the

thumb, then the ring finger. You pinched my little finger. You are squeezing my hand. You are squeezing the little finger." (Duration four minutes.)

Experiment 4.—Cross drawn on the hand: "Somebody moved my little finger. It opened up that cross you made. It makes a cut right through the center. Whatever you cut with the cross, that's open. You don't open the skin. Everything comes up higher and higher. It's in me. I feel a sensation that them lice are coming all the way to the top of the skin."

Experiment 5.—Two parallel lines drawn on the right hand: "You made a light cross. You moved toward Broadway (toward fingers). You're coming up around back to the wrist, back to center, straight across the hand (from thumb to little finger), coming up toward me. It's a line. You hesitate for a second. Now around the elbow. You are right in the elbow right now. I don't feel anything now." (Duration about four minutes.)

On the morning of January 24, the patient was clearer, oriented and made good contact. He stated: "The last thing I remember I was going to get a cup of coffee. I woke up yesterday and found myself in this bed. I remember what I said about lice but I don't think it's true."

Experiment 6.—Cross drawn on the right forearm: "Cross. Feels like you started moving crossways (along the lines). It's getting kind of normal again. I feel nothing." (Duration forty-five seconds.)

CASE 5.—Mo, aged 45, a laborer, a periodic drinker, was admitted on April 9, 1931, following a prolonged debauch. There developed an alcoholic delirium, marked pseudo-parkinsonism signs, and stupor with signs of wet brain. The patient died on April 20.

Experiment 1.—Impression made with a fingertip on the forehead: "Mark. A pip on my head (makes movements as if boring a hole into his forehead)."

Experiment 2.—Cross drawn on the forehead: "Every time I should remark it I feel it going up and down, like east and west (makes motion from the forehead outward). I feel the sting of the cut—angle shape."

Experiment 3.—Vertical line drawn on the hand: "A sore. Just when you cut that through it. It is sore from there on (demonstrates where cut was drawn from the knuckles of the second finger out along to the tip of the second finger). I feel a cut (points to forehead)."

Experiment 4.—Cross drawn on the forehead: "I feel a cross. I feel it every morning. I feel it too far about there (puts hand about 2 inches away from forehead). It is stinging in the center (points to approximately the intersection of the lines). It stings like an onion. When we peel an onion we cut it in half."

As in normal persons, lines tended to diffuse and spots disintegrated. Frequently, the changes are quicker and shifting is more definite (case 7, experiment 3). It is remarkable that in the nonpsychotic cases the perception of the after-effects of geometric figures is more apt to be of disintegrated portions of the figure, whereas in the hallucinating and unclear patients a total structure is perceived. The Gestalt may conform to the figure drawn or may form a structure suggested by the psychotic content (compare case 7, experiment 2, with case 6, experiment 1).

CASE 6.—Da, aged 38, a peddler, was admitted to the hospital with a picture of an alcoholic psychosis with delirium.

Experiment 1.—Cross drawn on the hand: "It's a star—Eucharistic."

CASE 7.—Cl, an engineer, aged 42, was admitted on April 11, 1931, in an agitated, apprehensive state. He showed signs of intoxication; rapid pulse, flushed face and dilated pupils. Within two days after admission he developed a vivid paranoid hallucinatory trend to which he reacted with marked agitation. The experiments were performed while the patient was relatively clear mentally.

Experiment 2.—Cross drawn on the left hand: "Cross whose center is farther toward the right. I feel the angles at the crossing. I feel the horizontal line better. Now they are faded somewhat. The perpendicular fades more rapidly than the horizontal. The horizontal I feel more intensely; deeper impression still; the flesh hasn't come out. . . . The shape now is two sides of an angle. The perpendicular line and the horizontal line which have faded less have come to make an angle. Now the horizontal line has moved up and has become the hypothenuse." (Duration four minutes.)

Experiment 3.—Circle drawn on the right hand: "A circle but not quite a round one. It is a disk. I feel the whole thing . . . a little more diffused. The intensity is less considerable. Has become less circular. Just a diffused mass of sensation. Still a sensation of warmth." (Active productions for three minutes.)

Experiment 4.—Triangle drawn on the left hand: "Triangle. Now it is becoming more of a plane figure. The sensation of the lines is a sensation of warmth. As the lines of the triangle fade slightly, the center makes it become a plane figure. It is a feeling of diffused warmth. The lines are like poles of an Indian wigwam; they crossed. The angles were distinct. I feel the warm sensation of both hands, fairly well diffused."

Experiment 5.—Impression made with the tip of a finger on the forehead: "Period. It has grown larger—general shape—it's wider—now it is almost round, big as a dime. Now it is very faint and vague."

CASE 8.—Si, a man, aged 42, who had been drinking until a few days previous to admission, had developed a paranoid hallucinatory state, and was agitated and apprehensive in response to vivid auditory and visual hallucinations.

Experiment 1.—Cross drawn on the hand: "I feel a cross on my hand. It is burning. It becomes a circle. It moves to the fingers. It's just a blot. It moves in a blot toward the thumb. It moves back to the fingers. Now it's moving in 'L' shape. Now it's a straight line to the fingers. Now a spot to the fingers to the right. . . ."

The angles of a triangle or cross were felt definitely by some patients. Indeed, the crossing point seemed to be utilized as a base from which activity originated (case 4, experiment 1). It was striking that the various joints of the hand and the portion of the extremities served as pivotal points to determine the direction of the movements or the location of the after-effects (case 4, experiments 1, 2 and 3, and case 8, experiment 1). These various points in the body stood in definite relationship to the movement tendencies of the after-effects, much more so than in normal subjects. One may say that the patients made use of the postural model of the body to orient themselves during the movements of the tactual sensations.

In several of the patients the tactual sensations and after-effects seemed to be below the surface of the skin. In some normal persons, as Schilder and I observed, sensation was perceived as being indented below the skin level or as passing through the tissue that had been touched. The patient in case 7, experiment 2, showed this tendency plainly. In case 5, experiments 1 and 2, this tendency was colored by the delirious content to mean an injury deep in the tissues. In the same patient (case 5, experiment 2) the after-effect was experienced on a level above, outside of the skin, a position that can be interpreted as being of the same order as the subdermal level, but the reverse. The patient in case 4 merged this experience with his visual and tactual hallucinations, forming a new tactual Gestalt (case 4, experiment 4) expressed in terms of his psychosis as insects crawling out of the skin "coming all the way to the top."

In the previously mentioned study, we noted tactual after-effects in a hypnotic subject arising from beneath the skin. As the patient expressed it: "It feels like dust coming out of the skin." These interesting observations can again be regarded as an intensification of a normal tendency to perceive tactual sensation as existing somewhere just below the skin. Probably, as Schilder suggested, this constitutes the level in the sensory model of the body for surface sensation, and not the actual surface of the skin. In everyday life, tactual sensations are localized on the surface of the skin with the aid of optic impressions; but under the conditions of cerebral intoxication perceptive changes appear and the sensation is localized under the skin.

Summary.—In alcoholic psychoses definite changes were found in sensation, the method of tactual after-effects being used. These changes are due to organic processes going on in the peripheral or central vestibular and tactual apparatus. The protocols presented point definitely to the conclusion that changed perception is the basis for the hallucination of the alcoholic patient. Without question, there are psychic factors that shape the hallucinations, but a definite basis exists in the physiopathology of the senses. The Heidelberg school, Beringer and Burger, came to a similar conclusion in that they found changes in perception of the lability of sensory thresholds.

EXPERIMENTAL RESULTS IN CASES OF SCHIZOPHRENIA

In order to ascertain whether the dissociative process in schizophrenia had any effect on tactual perception, a group of patients were chosen at random and the tactual after-effects were studied in the manner previously described. In working with this material one found difficulties due to negativism and lack of cooperation, resulting in diminished tactual productivity. Aside from this, the severity of the

psychosis seemed to have no influence qualitatively on the after-effects. The duration of the tactual phenomena in these patients was, on the average, two minutes—slightly less than was observed in normal subjects. The presence of tactual hallucinations in two patients, Si and Ha (a paranoid senile), did not influence the results in any way. In general, it can be said that the perceptual changes in the tactual after-effects in schizophrenia were not in any way distinctive for the psychosis.

The phenomena that occurred most commonly in the subjects were: When a single touch was made on the skin, the sensation gradually diffused and faded. When two simultaneous touches were made a short distance apart, there was a tendency for the after-effects to join together; with successive touches the tendency was to join together also, but both these reactions were varying. Lines perceived as a whole rapidly faded by diffusing, breaking into fragments or shriveling from the ends. Temperature sensations were associated often with tactual feelings, and occasionally pain was felt in the place of temperature. Lines, circles and geometric figures showed a tendency to slow movement and to diffusion and irregular effacement. In case 9, after a triangle was drawn, the sides disappeared while an irregular round area of touch appeared in the center of the triangle. The tendency to movement could be seen clearly in one case in which a portion of a line drawn was felt as a knob moving up and down along the line. In general, movement tendencies were slow and meager compared with those in other types of subjects. The crossing point of lines (in the cross) and the angles in geometric figures were more prominent in the after-effects here than was observed elsewhere. The protocols in several cases demonstrate this (see case 9). In normal persons the opposite situation prevailed; that is, angles and cross points rapidly faded out and could rarely be imagined or felt in the after-effects.

The perception of a drawn straight line as a wavy line with caterpillar-like undulations horizontally on the skin and into the air was a striking observation (experiments 3 and 5). In this case, the elevated phases of the line coincided with the points where the drawn line crossed the metacarpal bones. In the after-effects obtained following imagination of a tactual sensation, this impression of wavelike movement was even more marked. Further movements of the type described experimentally by Benussi[6] were observed by the same subject. Benussi noted that if two successive touches were made on the skin at short intervals, they felt as if joined by an arc moving through the air. It is highly possible that the phasic elements of the wavy line were developed from the sensation felt when crossing the metacarpal bones, combined with the Benussi tendency. The knuckles, or bones, served

6. Benussi, V.: Arch. f. d. ges. Psychol. **29**:385, 1913.

as points of fixation of the body image used in the development of the after-effects. In other words, various tactual tendencies were added to the basic tactual model (body image) of the body to form the perceived tactual complex in the after-effect. When visual images were present, and this was not frequent, they were rudimentary. Although the visual images were mixed with the tactual elements at the start, each series tended to develop in its own direction.

CASE 9.—Ei, a man, aged 31, who was brought to the hospital because of agitation and refusal to eat, gave a history of restlessness, irritability and feelings of weakness. In the hospital he was suspicious, complained of a "plot," and had auditory hallucinations and delusions of a homosexual nature; he was resistive and refused to eat. A diagnosis was made of paranoid dementia praecox. Soon after admission, the following results from tactual experimentation were obtained:

Experiment 2.—Line drawn on the dorsum of the hand: "I felt a line. It fades out by skipping every other spot. Now it concentrates in one spot. First

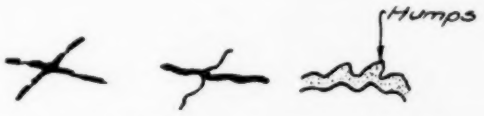

Fig. 1.—Tactual impressions in experiment 3, case 9.

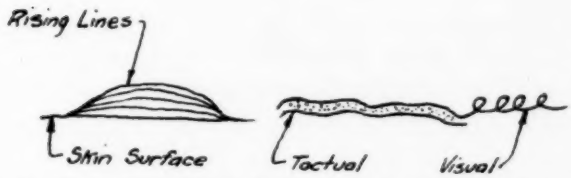

Fig. 2.—Tactual and visual impressions in experiment 5, case 9.

it was warm like tepid water running down the hand. Now it is cool and going away."

Experiment 3.—Cross drawn on the hand: "I felt a cross. I saw a cross green on a red background. I feel a prickling sensation. The lines are wavy. The vertical line fades. I feel the cross point strongly. The humps are where you crossed the bone. Now the horizontal line takes a broader field. The pricking sensation is gone; only a faint feeling of touch left."

Experiment 4.—Two simultaneous touches 1½ inches apart: "I felt a dull pressure all in one touch, it spreads out. It is a light pain. It expands and is wavy somewhat along it. Part of the line disappears and two points remain. They are intensified."

Experiment 5.—Patient asked to imagine a line drawn on the hand: "I feel a line. I feel half-circles rising into the air on it. I don't see anything. The surface lines become a line in the air. I feel a single wavy line. I have the vision of spirals coming off the wavy line. The line felt as if it rose itself into the air."

Comment.—In schizophrenia there are no basic changes in the physiology of after-effects of tactual perception. Whatever deviations

from the normal may be present are due to particular elaborations associated with schizophrenic thought. It is possible that the psychology of schizophrenia tends toward a special interest in angles and crossing points, and that this interest prevents the fading of these points in the after-effects. It is likewise probable that one deals in this problem with slight changes in the sensory sphere proper (the perceptual apparatus). The observations show distinctly that schizophrenic hallucinations must have a mechanism other than do the hallucinations of alcoholic patients, and that schizophrenic hallucinations are less dependent on the immediate changes in the sensory apparatus. Beringer,[7] using the technic of Weizäcker and Stein, found analogies between the reaction of normal exhausted persons and those with schizophrenia. According to Beringer, there is no real disturbance of function in sensory perception, but a disturbance in integration which can be likened to that produced by fatigue.

It is not known yet in what way psychic factors influence tactual after-effects. Previous experiments on a hypnotic patient showed that there can be a considerable change through psychic influence. The elements contained in the tactual after-effects are dependent not only on what is in the perceptual field, but on all that goes on in the individual. At any rate, in the field of after-effects and eidetic images, the organic physiologic factors mingle with the psychic factors. From this point of view it is worth while to discuss the results in a manic patient who had the feeling at the outset of his psychosis that everything was moving fast in his surroundings.

In this patient, the representations and after-effects were vivid and of relatively long duration. In fact, the rapidly appearing representations of previous sensations during the course of a series of after-effects gave the tactual picture something of a flighty character. The lines drawn were felt as changing constantly because of fadings and effacement. Lines and spots showed a definite disposition toward movement; frequently it was rotary or a continuation of the direction implied in the line or figure (case 10, figs. 4 and 3). The same tendency for lines to move into the air with something resembling a wave motion was observed here as before (experiment 3). Even the tendency to multiplicity of images was observed here (experiment 2).

In this patient, as in the normals, angles and crossing-points were felt with difficulty or not at all. When two simultaneous spots were made on the skin, the tendency for their after-effects to coalesce was present, whereas with successive spots, one tended to displace the other.

7. Beringer, K.: Arch. f. Psychiat. **92**:476, 1930.

CASE 10.—Li, a designer, aged 21, was admitted with the history that while in a theater he got on the stage and attempted to give an impersonation of Napoleon. On admission, he was talkative, euphoric, elated, flighty and overactive. He gave a rambling account of his actions, and was distractible, grandiose and manic. The patient said: "I got on the stage because the show was unusual. A girl sat next to me, a perfect lady, the girl of my dreams. Soon I saw a lot of people lined up against the wall. I had a feeling something was going to happen. . . . Everybody seemed to be so busy. Everybody was getting up and changing their seats. I thought I would get up and experiment too, so I changed my seat and then I realized the whole thing was really a party. The whole thing was beautiful and I felt quite happy. . . . I felt I was in the midst of a party and had to do my share . . . that's why I got up on the stage to imitate Washington or Napoleon."

Experiment 1.—Vertical line drawn on the hand: "Straight line. You are going into my skin, you are pressing. Sort of a tingling feeling. I feel it nearer to me. Seems to be getting stronger. It is a straight line moving toward me; toward you it's weak. It seems to be getting into a spot. Feels somewhat like a worm, twisting around like a wheel. It remains about the same. It is still moving. My mind comes back to the first sensation but what I feel is different. The spot represents the end of the line."

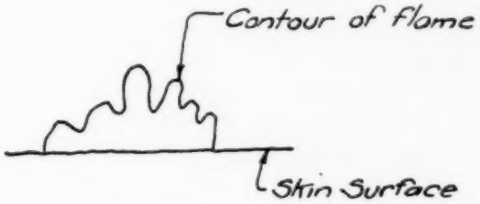

Fig. 3.—Tactual and visual impressions in experiment 3, case 10.

Experiment 2.—Cross on the left hand: "Cross. The horizontal line is the stronger of the two. I don't actually feel any sensation where the lines cross. One line feels higher than the other. I feel the line you have drawn across will go infinite; I don't know where. It is moving vertically. The cross does not mean anything. I can see it but I don't feel it. There is only one line. I feel on the left hand a whole lot of lines crossing."

Experiment 3.—Circle on the right hand: "Feel a circle. Now I feel the one on my left hand going in a counter-clockwise way. It reminds me of a worm. The left is stronger than the right. On the right hand I can feel the three lines you drew yesterday and the circle you made today very clearly. The diameter of the circle is about 1 inch. The line in thickness seems to remain the same. It is tingling; the left more than the right. I feel it moving upwards like fire." (When asked to explain the fire sensation): "I see it as if my eyes were in the same plane as my hand looking sidewise. The outline of the patch is very irregular. When I think about it, I look for an imaginary point. On the right hand I feel the irregular border."

Experiment 4.—Two spots made simultaneously on the left hand: "Two dots. They seem to be joining, going together. They both move at an equal distance. I feel one spot. It's growing larger, more intense, much stronger. It's got that fire feeling of the sun. On the right hand is the same circle and triangle. You

make these on my hand and they seem to be made forever. It remains the same, but it seems to be going around, just as though it were spinning around like a top."

SUMMARY

1. Tactual perception in sixteen patients with alcoholic psychoses, ten with schizophrenic psychoses and one senile and one manic patient were studied during the active phases of the psychoses by the method of examining the tactual after-effects.

2. In alcoholic conditions the duration of the after-effects was prolonged and the improvement in the alcoholic intoxication was paralleled by a decrease in the duration, intensity and elaborateness of the after-images.

3. Sensations of temperature were present and at times were perceived as pain.

4. The type of alcoholic condition made no essential difference as to the nature or intensity of the after-effects.

5. Rapid diminution and multiplication of spots of sensation, rapid rotary movements, fragmentation of lines, and diffusion, fading and disintegration of tactual impressions were perceived by the patient in terms of his mental content.

6. The disposition toward disintegration of tactual perception in alcoholic persons is analogous to the disintegrating effect on tactual perception of vestibular irritation, as was shown in the study of normal persons.

7. Two simultaneous spots showed a tendency to coalesce when their tactual images were retained; at times they were perceived together initially.

8. Persons addicted to alcoholism showed an inclination to perceive after-effects of geometric figures as a total structure (Gestalt).

9. Tactual experiences were often referred to pivotal parts of the body, such as knuckles, arms and fingers, which have some relation to the postural model of the body.

10. Sensations were often perceived below the skin level and were associated with movements from that level upward into the air.

11. Schizophrenic patients showed no qualitative change in the perception of after-effects as compared with the normal. Effacements, diffusion, fading, joining of two separate points, temperature sensations, movement tendencies, etc., were all present in these patients, but were diminished in degree and rapidity as compared with the normal.

12. Cross-points and angles in geometric figures were noticeably present in the after-images in patients in the schizophrenic group.

13. The Benussi phenomenon was observed in one patient.

14. Wavy lines, extending into the air or horizontally, with the rising phase of the waved line corresponding to bones crossed by the experimenter's finger, were observed.

15. The experimental results were not influenced by the presence in two patients of tactual hallucinations.

CONCLUSIONS

Studies of the after-effects of tactual sensations made on patients with alcoholic and schizophrenic psychoses showed that the tendencies toward change in the after-effects found in normal persons were present in the patients. In persons suffering from alcoholism the after-effects change in such a manner as to indicate that there are organic changes in the perceptive field. These organic changes may be in the nature of sensory or vestibular influences. In alcoholism, changed sensory perception is basic for the existence of hallucinations. In patients with schizophrenia, there are no changes in the perceptive field; hallucinations among these patients are related to the special thought processes of schizophrenia. It is still an open question in what way these special thought processes influence secondarily the perceptual field.

INCONSTANCY OF THE FORMAL STRUCTURE OF THE PERSONALITY

EXPERIMENTAL STUDY OF THE INFLUENCE OF MESCALINE ON THE RORSCHACH TEST

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The drug mescaline [1] has a peculiar influence on the central nervous system of man.[2] Within a brief time after its introduction into the organism it causes a clearcut disorder. The most characteristic symptoms are visual phenomena. The subject sees, with closed eyes, with open eyes in a dark room or against the background of a screen, a wealth of visons characterized by lavish colors and more or less geometric forms, all of which are in perpetual movement.

Among other symptoms, some of which are remarkably constant and occur in practically every case, are: a general passive attitude; a surprising disorientation as to the time that elapses during the experiment; a tendency to observe tiny details in the immediate environment, such as beginning fine cracks in the wall; an increased sensitiveness and attention to all colors in the environment; a feeling that the whole body is "lighter"; a pleasant attitude of giving in entirely to the enjoyment of the visions, with absence of any feelings of anxiety; a certain amount of euphoria, independent of the visions (one of our subjects had a tendency to laugh without reason before any visions had appeared); a certain amount of lucidity and objectivity with regard to the abnormal experiences, and an inability to conjure up exactly in color and form any desired image. The exact shade and form of the visions are usually remembered for a very long time.

Physically, there is slowing of the pulse, the pupils become dilated, and there is frequently an initial feeling of malaise which may go as far as nausea. The general picture of this reaction to mescaline is remark-

1. In this study the mescaline was administered in the form of mescaline sulphate.

2. For literature, see: Beringer: Der Meskalinrausch, Berlin, Julius Springer, 1927. Rouhier: La plante qui fait les yeux émerveilées. Le peyotl, Paris, Gaston Doin, 1927. Klüver: Mescal Visions and Eidetic Vision, Am. J. Psychol. **37**:502, 1926. Pharmacologic information was furnished by Dr. Gudernatsch of the Hoffmann-La Roche Chemical Works, New York.

ably similar in all subjects. With larger doses, a more profound effect with peculiar distortion of bodily sensations and other phenomena may be produced.

In this series of experiments with mescaline intoxication on normal subjects, the effects of mescaline were true to type. The visual phenomena were the most striking feature. In his retrospective account, one subject stated that all the visions had the following characteristics in common:

> They moved very quickly. The colors were alive, with a quality beyond brilliance or movement. They had a vitality of their own. The visions were usually imposed one on the other. I saw from two to five planes at once, all distinct but all at different distances and in different positions. They had the reality of pictures by some one else. They were palpably present, but unlike dreams or reveries they bore (with a few exceptions) no relation to anything I know. Unlike experiences under ether, I was able to detach myself from them at any moment.

A good impression of these optic phenomena is given by the attempt of one subject to paint in oil a few of the scenes on the day after his mescaline test. He painted four pictures. Since it is very difficult to gain a clear realization of these visual experiences in words, and since mescaline hallucinations are of considerable psychopathologic interest, two of these paintings are given here as illustrations (figs. 1 and 2). He wrote of these paintings in his retrospective account:

> . . . A field of century plants. I have painted only one plane, but there were actually five at the same time. This is the only vision that had any apparent connection with the drug (century plants, pulque, also called mescal). The plants were in sandy fields and did not move in relation to their background, though all five planes moved separately in different directions and at different angles from the eye. (Fig. 1.)
>
> The second vision was seen while the physician played the phonograph. The background was flames. The black figures moved up black stairways. Their movements were angular and mechanical. In this case there was one background, but the stairs were, like the century plants, at different distances from me. (Fig. 2.)

There is only one feature of the mescaline psychosis that is not sufficiently brought out in the literature, and on which our experiments throw new light, and that is concerned with the after-effects. It is usually stated that after the intoxication no effects remain except a certain acuity of vision and sensitiveness or increased attention to colors. It seems, however, that even days after the intoxication an after-effect may still be demonstrated in dreams. One subject had four subsequent dreams, in each of which he had the feeling that there was a strong light in front of him or above him. Twice he awoke and reached to turn off his bedside lamp. As he reached he woke up, and realized that the light was not turned on at all. He was quite sure that when he tried to turn it out he was not fully awake. Each of the dreams

Fig. 1.—Painting of mescaline vision.

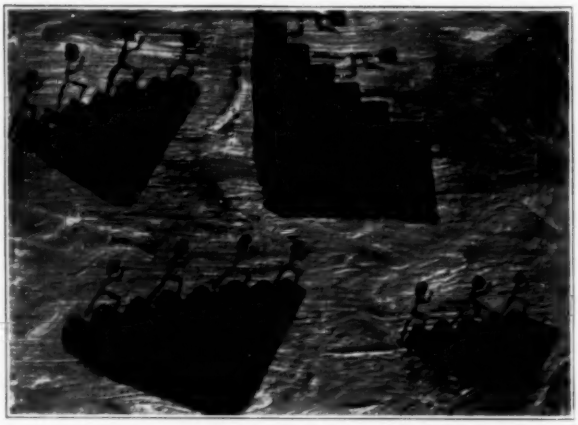

Fig. 2.—Painting of mescaline vision.

came about five minutes after falling asleep. We believe that this influence of mescaline on subsequent dreams is of considerable importance for the psychology of dreams.

Most investigators who have paid attention to the point, Beringer, for example, have not found that different types of people showed correspondingly different types of behavior under mescaline Only Bensheim, in a recent paper, thought that he could demonstrate such differences in personality types. But his observations do not seem to be entirely conclusive, quite apart from the fact that they were very summarily reported.[3]

In our experiments with mescaline intoxication on normal subjects the reaction to mescaline as a whole followed its typical course. We were struck, however, by some individual differences in the reactions of different people. These differences seemed to point to certain peculiarities of personality of the subjects. It can happen, for example, that subjects show a tendency to misinterpret the behavior of those who make observations and conduct the experiment, resenting the fact that the observer disturbs quiet enjoyment of the visions by questions, etc. They may feel that the observer is definitely inconsiderate, or, if they have the desire to express in words all that they see, they may feel that if the observer does not listen to them all the time he does not conduct the experiment with enough attention to details. This behavior may be sufficiently pronounced to warrant the description of a transient paranoid attitude with a good deal of irritation and resentment.

One of the subjects of Prentiss and Morgan showed during the experiment a certain resentment against the observers. He felt that under the influence of mescal he was in a condition of "mental inferiority," and that the observers enjoyed seeing him in such a state. One of our subjects showed a similar peculiar affective attitude during the experiment. He corrected his ideas afterward to a large extent, but not entirely. For some time afterward he felt that the experiment had not been conducted with adequate scientific precautions, and that he might have died during the experiment because sufficient attention was not paid to his physical status. He seemed to forget that he had spent most of the time in an obvious state of enjoyment, with a tendency to overtalkativeness and exhilaration, and complained afterward that he felt miserable and neglected all the time.

Several such experiences suggested that the mescaline experiment might reveal certain aspects of the personality usually not so apparent. It seemed interesting, therefore, to determine with some suitable method whether diagnoses of personality could be made during mescaline intoxication. Such attempts were made with the Rorschach test. The possible

3. Wertham, Frederic: Progress in Psychiatry. IV. Experimental Type Psychology, Arch. Neurol. & Psychiat. **24**:605 (Sept.) 1930.

effect of mescal on the Rorschach test seemed interesting also because this test is based entirely on visual perceptions and interpretations. The reaction of a subject could not be predicted if this test was performed during an experiment in which his psychovisual attitude was changed as profoundly as it is under mescaline.

The Rorschach test [4] consists of a series of ten meaningless ink-blot pictures, partly in colors. The subject is given these pictures and asked to tell what forms he sees. These answers are evaluated according to definite categories which will be indicated later. This test is not an exact method. The subjective interpretation of the one who evaluates the test cannot be entirely ruled out. Its conclusions are based entirely on empirical data, about which those experienced in using the test do not entirely agree. But with these reservations the test is extremely useful, and has yielded surprisingly correct formulations of personality.

TECHNIC OF THE RORSCHACH TEST

The ten ink-blot figures of which the test consists are shown to the subject in a definite order. He is told only to tell what he sees, and he may give as many answers for any one figure as he wishes. Each answer is recorded and classified according to a formula. One cannot rely on these formulas alone, however, but must always consider the answers themselves.

One of the most essential points in the interpretation is the classification of the answers according to whether they are dynamic, form or color answers. Most of the answers are form answers (*F*), which are given in consideration of the form alone, while the color is neglected and there is no dynamic element involved. According to the experience of the frequency of certain answers in intelligent and unintelligent people, the *F* answers are differentiated into well seen (*F+*) and poorly seen (*F—*) answers. The average percentage of *F+* answers in relation to all *F* answers for an intellectual person is from 80 to 90 per cent. As a rule, the higher the intelligence, the higher is the *F+* percentage, but this is true only in general. The optimum of *F+* percentage is not 100. Pedantic and overly self-conscious natures, as well as persons in a depression, may give 100 per cent *F+* answers. Likewise, the *F+* percentage is higher when the intelligence is more directed toward logical and well controlled thinking than toward originality and production of new ideas.

"Blood" as an answer to designate a red spot is a mere color (*C*) answer. "Butterfly" for the red central spot on picture III of the test is an answer due to both form and color perception, therefore an *FC* answer. "Inkspot" for the lateral blue areas of picture x of the test relates more to the color than to the form, and is therefore a color-form answer (*CF*). As a whole, colors indicate extravert affectivity. A tendency to react promptly, directly and in relation to the expression of mood in other persons is represented by the color answers. Color answers are therefore to a certain extent an indicator of a syntonic temperament. However, a depressed person very rarely sees colors.

C, *CF* and *FC* answers do not have the same significance. *C* answers represent an uncontrolled impulsiveness. They occur in a majority of subjects with temper tantrums, crying spells, etc. The *FC* answers, on the other hand, indicate simply

4. Rorschach, Hermann: Psychodiagnostik, Bern, E. Bircher, 1921.

a good affective contact with the environment, social qualities, pleasure in meeting people and a good affective rapport. The rôle of the *CF* answers is an intermediate one.

In addition to the *F* and *C* answers, there are dynamic answers (*dyn*). In spite of the fact that *dyn* answers are perhaps the most important factor in the test, they are often misunderstood. Not every answer that expresses a movement is a dynamic answer. Only those answers expressing motion are *dyn* that show a projection of the subject into the answer. The subject may express this frequently by gestures. He often expresses or feels an emotion with the movement. The essential character of the *dyn* answer is illustrated when a person sees in a cloud now the form of Italy, now a man stretching as though tired. The answer "a person sitting as though dejected" may be *dyn*, though there is no actual movement in it. As a whole, the *dyn* answers indicate introvert affectivity. The subject may repress his immediate reaction to the figure into the unconscious, so that its affect is delayed or indirect. The *dyn* answers represent schizoid affectivity within normal limits. They are also dependent on the intelligence. A feebleminded person does not give *dyn* answers. An intelligent extravert may give quite a few of them.

The number of *dyn* answers must be compared with the number of color replies. This relationship gives a picture of the relationship between schizoid and syntonic affectivity. Many kinds of interfering circumstances may change the ratio between them. Depressed persons do not dare to give *dyn* replies. Introverts who try to become extraverts or to hide their introversion in their way of living may repress the *dyn* answers unconsciously. The examiner can discover such a repression in a test without *dyn* answers by noticing that many other factors are well represented that usually are proportional to the *dyn* answers. Such factors are well seen original answers, few answers in terms of animals, etc. As far as intelligence is concerned, a high rating in *dyn* does not alone indicate a high intelligence. It denotes certain characteristics of the intelligence, such as originality, the ability and the ambition to work things out intellectually, a certain pleasure in doing intellectual work or a creative ability.

A special kind of form answers are those that concern the white portions surrounded by the colored figure ("white" answers). A high number of them indicates negativism or, in dynamic personalities, great self-critique.

The second important classification of answers concerns the seeing of the whole figure or a detail of it. The number of well seen answers concerning the whole figure (*W*) is usually higher in intelligent people. Feebleminded persons rarely have well seen *W* answers. In the sphere of the intelligence, the *W* answers are indicative of a tendency to summarize and to develop impressions. They are usually proportional to the *dyn* answers. But even a person without dynamic qualities and without originality may give many well seen *W* if he has great intellectual ambition. Some *W* are formed by abstraction and others by combination. Many abstractive *W*, together with other signs of high intelligence, indicate that the intelligence is directed more particularly toward scientific and philosophic problems. Combinative *W* are more characteristic of artistic interests and abilities.

Answers that refer to natural subdivisions of the figures are called detail answers (*D*). Answers considering more or less artificially separated small parts are small detail answers (*Dd*). Many well-seen *Dd* are suggestive of good observation. In the test of a natural scientist, *D* and *Dd* may occur more frequently than in that of a philosopher. A person who has both many *Dd* and many *W* in his test, but who neglects the *D*, is usually impractical.

The third important consideration in evaluating answers has to do with their content. The chief factor in the content is the percentage of animal answers (*A* per cent) in respect to the total number of answers. This proportion indicates a tendency to stereotypy in thinking. The *A* per cent is high in unintelligent and unproductive people. If it is high in an intelligent person it means that his intelligence is probably more reproductive than creative. The *A* per cent is higher in old age than in youth.

A further consideration of the content refers to the number of anatomic answers. In nonprofessional persons, a high number of anatomic answers usually indicates an "intelligence complex."

The relationship of parts of animals to animals, and parts of men to men, has a significance similar to the relationship of *D* to *W*. If many objects are seen, it may suggest good practical qualities.

Finally, the answers are classified according to their frequency. Original answers, known as *O*, are answers that occur less frequently than once in 100 normal people. The relationship of *O* to all answers (*O* per cent) and the absolute number of *O*+ answers are important factors. The *O* per cent is in inverse proportion to the *A* per cent. A high *O* per cent indicates original, creative, rather dissociated thinking. In many autistic subjects the percentage is too high. It is also high in many artists. The total number of *O*+ answers is taken into consideration in reckoning intelligence. An intelligent person is supposed to have quite a few *O*+ answers. The opposite of the original answers are the common answers (*Co*). They represent good common sense and a satisfactory intellectual rapport with other people.

There are many other facts to be given consideration by the examiner. For example, a repressed person may give dynamic answers and color answers only as the last answers in his reactions to the figures. A person who is accustomed to very logical thinking may regularly give first a *W* answer, then a *D*, then *Dd*, etc.

OUR PROCEDURE

In our experiments we used the following procedure: One of us (Dr. Wertham) conducted the mescaline experiments. He also obtained a protocol of the Rorschach test a considerable time before the day of the mescaline experiment. Then, while the subject was under the influence of the drug, he obtained another Rorschach test protocol. It was found that the two Rorschach tests, the first one and the one obtained when the subject was under the influence of mescaline, may be remarkably similar. In one case, that of the subject who painted his "visions" afterward, the two protocols were almost identical. In some cases there were slight differences that did not greatly affect the interpretation as a whole.

In the one case, however, that forms the basis of this report, there was a remarkable difference. The protocols in this case, with some others, were sent, numbered, to the other experimenter (Dr. Bleuler), who did not know under what circumstances the protocols were obtained. He did not even know that there was a possibility that two protocols might come from the same person. The only data available to him were the age, sex and occupation of the subjects. It should be added

that the one who conducted the mescaline experiments also obtained complete personality data both from the subjects themselves and as far as possible from objective sources. Nothing was known about these data by the experimenter who interpreted the protocols.

Some remarks are necessary about the taking of the Rorschach test with a subject under the influence of mescaline. The test was given while the subjects were very much preoccupied with their mescaline visions, and it required a good deal of persistence to get their attention on the test and to hold it there. The subjects were apt to compare the Rorschach blot picture with the visions they could obtain by closing their eyes, and they would express the opinion that the mescaline visions were infinitely more interesting and more beautiful than any pictures the test could offer. For this reason the test under mescaline lasted longer than it did when given under ordinary circumstances. Special attention was paid to the fact that the subjects should feel that the Rorschach test was only an incidental feature of the experiment. It was relatively easy to obtain this effect, because a number of other tests (association test, investigation of eidetic phenomena, etc.) were carried out during the same period.

Numerous observers have found that when the Rorschach test is repeated, the protocols are usually remarkably similar. For this reason it seemed of considerable interest that in one case the two protocols were so different that the one of us who has a great deal of experience with the Rorschach test,[5] especially the phase of it that refers to the similarities and differences in different people, without any doubt or hesitation considered the two protocols as belonging to two different people with distinct and clearcut personality patterns. The problems suggested by this singular occurrence and the fact that some conclusions of general psychopathologic interest may be drawn from it warrant the presentation of the material of this experiment.

REPORT OF A CASE

About forty minutes after the injection of the mescaline (mescaline sulphate [0.3 Gm.] injected intramuscularly), the visions began. The subject lay with closed eyes and saw successions of brightly colored images, for example, pyramids leading up to glistening bubbles against a blue background. He felt that he could look into enormous distance. The visions occurred in patterns which folded and unfolded in brilliant colors. He saw, or expressed, these patterns in objects and scenes that contained geometric patterns; e. g., thousands and thousands of peacocks, whole rows of trees with insects' wings for leaves through which the sun shone or ships sinking in a blue sea, with great waves rushing symmetrically in, curling like sea shells in marvelous cascades of color.

5. Bleuler, M.: Der Rorschach'sche Formdeutversuch bei Geschwistern, Dissertation, Zurich, 1928.

Some visions came from the right, as if they were on a belt, and others from the left. The subject felt it impossible to describe the variety of images. He felt that he could visualize almost any scene, but when he tried he found that the image that appeared after a time was different both in shape and color. Later, he could visualize any desired color but not a desired form. Trying to see a pink flower, the image of a pink battleship would appear. Efforts to conjure up an erotic scene were unsuccessful. The scenes shifted continuously, and the visions followed one another in riotous profusion. The shapes were always regular and geometric. As an example: "The scene was one of incredible magnificence. Nebulae and stars moved in circular and elliptic orbits. The stars were gold points, the space was dark blue, and the nebulae were orange. I felt that by an effort I could see into the very center of the universe. I made the effort, and the stars and nebulae streamed by me as I looked farther and farther into space."

There was sometimes a tendency for the subject to project himself into his vision. He said, for instance, "I see mosaics in small tiles. It is a room, the corners of which are rounded and nicely curved. It looked as if I were in the room. I could almost see myself."

In the dark room the visions occurred while he had his eyes open; but there no intense colors appeared. He saw hundreds of very formal patterns, but all in gray or neutral hues.

While the subject lay on the couch with closed eyes, attentive to his visions, one could observe that he seemed very happy. He said, "I want to laugh (he did so), but I don't know why." He enjoyed his visions, and disliked being interrupted by questions and tests. All the scenes he saw were novel ("nothing I ever saw before or hope to see again"). He paid no attention to the observers. "You all seem so far away. You are not in my world. I have a world of my own." Disorientation in time was very marked. After the experiment had lasted about two and a half hours, he gave the time as four hours later than it actually was.

As a feature rather unusual in reactions to mescaline, but one of considerable psychopathologic significance, it should be noted that on three occasions definite auditory phenomena appeared. The subject described them at the time and later recorded them in his retrospective account as definite facts. While the conductor of the experiment was away from the room, the subject visualized him standing in a characteristic way, with papers in his hand, and plainly heard him talking to some one whom the subject could not see. The subject saw the exact place in front of a door on the corridor where the conversation took place. In reality, there was no such conversation. Later, the subject visualized gorgeous tapestries with austere and regal designs floating slowly upward to the sound of music. The music was distinctly audible to him, and he accounted for it by the theory that a gramophone was being played in a neighboring room. The tapestries as they moved kept exact time to the music. No such music was actually played. Toward the latter part of the intoxication some of the elements of the pictures were still in motion, but the majority were stationary. The visions were still distinct, but each one was destroyed by the explosion of a bomb. "The scene would form, then a bomb would gradually fill it until it was entirely blotted out. At the moment of the scene's eclipse, the bomb would explode with an audible pop, and a muscular jerk would cause me to open my eyes."

In his retrospective account, the subject remembered clearly the details of his visions and could formulate them exactly, that is to say, as exactly as he could during the time of the intoxication. On the day after the experiment, he felt mentally and physically in a somewhat depressed state.

The Rorschach test given when the subject was under the influence of mescaline gave the following results:

Total number of answers	25	Well seen form answers	16
Whole answers	8	Poorly seen form answers	2
Detail answers	11	Dynamic answers	5
Small detail answers	5	Form-color answers	2
Small detail white answers	1	(Color was also slightly indicated in another answer)	
		Color-form and color answers	0
		Relation of dynamic to color answers, 5 to	1
		Answers in which black is considered as a color occurred several times	
Animal answers	5	Well seen original answers	3
Animal detail answers	5	Poorly seen original answers	2
Human form answers	5	Common answers	5
Human form detail answers	3	Percentage of well seen form answers	89
Geographic (map) answers	3	Percentage of animal answers	40
Clouds	2	Percentage of original answers	20
Figure	1	Percentage of common answers	20
Object	1		

INTERPRETATION

The majority of the individual answers are clear, well seen and not too simple. Exceptionally original or especially sharply observed answers are almost entirely lacking. The number of whole answers and the percentage of well seen form answers and of original and common answers all indicate a good average intelligence. There are a few more dynamic answers than one would expect from the other intelligence factors, and at the same time it is conspicuous that the number of original answers is somewhat smaller. (In general, dynamic answers and well seen original answers increase proportionately in number.) These observations permit the conclusion that the intelligence of the subject as a whole is above the general average, but hardly above the average of "intellectuals."

There is no evident leaning toward a special direction of thought, such as history of art, anatomy or pathology. The whole answers not only are given abstractly but indicate marked ability for combination. The relationship of whole to detail is about average. The high percentage of common answers is striking and indicates a good intellectual adaptability. The answers seem to have been given quickly, without special caution, as is indicated, for instance, by the two poorly seen form answers. The fact that among the color answers only form-color answers are represented would agree with the interpretation of a good combinatory ability. The relatively high number of dynamic answers indicates in general the inclination and the ability to elaborate associations intrapsychically, to divide them into their elements and to derive from them new creative concepts. The result of this ability, however, the well seen original answers, are rare, an important contrast that will

find its parallel in our discussion of affectivity. The succession of whole, detail and small detail answers in each picture shows nothing remarkable. The relation of animal and human form answers to animal and human detail answers corresponds to the relationship between whole and detail answers. Anatomic answers are lacking, while there is a predilection for seeing maps and figures.

One may say, therefore, that the subject is a clear thinker, who has well adapted himself to current ways of thinking. There are no "mania" for originality and no exaggerated intellectual fervor in any special field. But there are will and ability to high average intellectual work. No abstract philosophical or purely theoretical interest above the average exists; rather is there enjoyment of good form and of elaboration and artistic interests. Under the surface there are a far-reaching intrapsychic elaboration of impressions, and also definite intellectual creative abilities. The latter qualities, however, are not sufficiently cultivated and brought to fruition.

The strong predominance of dynamic answers over color answers indicates a more introverted than extraverted affectivity, although form-color answers are also represented. There exists, therefore, despite the tendency to a schizoid general personality, a good affective adaptation and a good social manner. Since there are a high percentage of common answers and not an especially high percentage of original answers, and since the subject never evades the usual answer and does not allow himself any peculiarities whatsoever, one can deduce that the schizoid traits recede in ordinary life behind pronounced sociable tendencies. Adding to this the lack of colors, one may further deduce that his emotional attitude is even, and that he does not let himself be impelled by momentary impressions, but makes use of single impressions gained over a longer period of time. There is also a tendency to have simultaneous conflicting emotional attitudes.

Of great interest are the answers in which black as a color, and the different shades within the black, are considered. It is most likely that these express some sort of passive suffering hidden under the surface. The subject suffers from something without resisting ("Weltschmerz"). The content of the dynamic answers points in the same direction. The figures seen do not fight or dance, nor are they poised for action; they are passive, staring at each other and placid, and have drooping arms. Not even picture IX, usually interpreted as two brown figures fighting with each other, was an exception. There is therefore a parallelism between affectivity and intelligence. The subject hides his personal and original world of emotions under an impersonal, somewhat smooth sociability, whereby a dull, unclear sensation of pain is not lacking. In the same way, he hides his creative intellectual intelligence under a good adaptation to the average ways of thinking.

The Rorschach test taken under normal conditions some time before the mescaline experiment gave the following results:

Total answers	60	Well seen form answers	51
Whole answers	3	Poorly seen form answers	3
Detail answers	17	Dynamic answers (2 of which in small detail answers)	3
Small detail answers	27	Form-color answers	3
Details seen where usually whole figure is seen	4	Color-form answers and color answers	0
Detail white answers	1	Relationship of dynamic to color answers, 3 to	1.5
Small detail white answers	8		
Animal answers	4	Well seen original answers	17
Animal detail answers	8	Poorly seen original answers	1
Human form answers	4	Common answers	3
Human form detail answers	27	Percentage of well seen form answers	94
Geographic (map) answers	7	Percentage of well seen animal answers	20
Anatomic answers	3	Percentage of well seen original answers	20
Objects	3	Percentage of common answers	5
Plants	2		
Answers with other content	2		

INTERPRETATION

This is a very peculiar and characteristic protocol, of which the most conspicuous traits are the pronounced small detail type of answer, with many "white answers," and the frequent answers in which only one small part of the body is given while most people usually see a whole figure. It is further characteristic that there are two dynamic answers in small detail interpretations. These, in contrast to their evaluation by Behn-Eschenburg and Usteri, are taken as having the same significance as other dynamic answers. Such answers are rare, and one may go over many protocols without encountering one. Furthermore, a strong perseveration in seeing small profiles is conspicuous. It must be noted that they are apparently sharply and exactly seen. One may even say that the empathy in the expression and position of the faces is usually so fine that the significance of these profiles approaches that of the dynamic answers. A further indication that one deals with a dynamic type is the fact that the dynamic answers are not given where they seem most obvious, namely, in the earlier pictures of the test, but only in the later pictures. This indicates repressions, a suppression of schizoid traits in consciousness.

The subject's personality is, therefore, a strongly introverted one. To this description must be added distrust of his own originality and of his own intellectual work, exaggerated self-critique, an anxious and hesitant type of thinking and a certain lack of free expression of impulses, of simplicity, of naturalness and of primitive direct emotions. There seems to be a slight depression, far within the limits of the normal. These traits are interpreted from the many white answers, the many answers in which a part of the body is given alone instead of as a part of the usual whole figure, the lack of whole answers and the enormous preponderance of small detail answers, the pronounced

prevalence of human and animal details over whole human and animal figures, the long reaction time, the narrow range of answers in comparison with their high number, etc.

The high number of original and very clearly seen forms indicates an intelligence far above the average, probably also far above the average for "intellectuals." It is far better also than that of the other subjects (among whom the same person, under the influence of mescaline, was included, unknown to the interpreter of the test). There is a pronounced tendency to elaborate and to analyze impressions intellectually, even when it is not necessary for practical reasons. There is a natural impulse to thinking and investigating. The subject is a keen and excellent observer and a worker in minute details. The most unimportant details do not escape him. He notes them even when they seem to have no practical significance whatsoever. In his thinking he expresses extreme self control and makes a practice of suppressing ideas of which he is not absolutely certain. The ability and interest in seeing the essential, in forming a well rounded picture from his observations and in synthesizing are not well developed in comparison to the rest of the intelligence. Independence and originality are not lacking, but their action is considerably impaired by the much too rigid conscious self-control. This can be deduced from the preponderance of small detail over whole answers and of human and animal detail over human and animal form answers, the noting of small parts of the body alone when the whole figure is usually seen, the white answers, etc.

The general picture of the personality is one of a schizoid professional man concerned with small detail work, most likely with natural science interests. The personality is well adapted, as can be seen from the three form-color answers, the sympathetic understanding of the expression of the tiny profiles, the absence of abstract answers and the not too high percentage of original answers. Although the subject seems to be quite sociable, there is a lack of free emotional life, natural unrestraint and passionate devotion. On the other hand, little details have an influence indirectly and over a long time. For example, the subject may feel hurt and react to a small offense only years after it has happened.

COMMENT

The interpretations were made when the interpreter did not know that both the protocols given belonged to the same person. They show a remarkable contrast between these two "personalities." In a further critical comparison, however, based on the knowledge that the two protocols belong to one person, one has to consider the absolute number of answers—there were 25 in the first protocol and 60 in the second. It is well known that this absolute number of answers is open to chance influences and may vary in the same subject. For purposes of comparison of protocols in which the absolute number is very different, one

uses a procedure described by Rorschach. Instead of comparing the two original protocols, one reduces the length of the one with many answers by considering only the first three answers to each picture. In this way the second protocol of our subject takes on the following form:

Total answers	29	Well seen form answers	23
Whole answers	3	Poorly seen form answers	3
Detail answers	11	Dynamic answers	1
Small detail answers	8	Form-color answers	2
Small parts of the body where whole figure usually seen	3	Relation of dynamic to color answers, 1 to	1
Detail white answers	1		
Small detail white answers	3		
Animal answers	4	Well seen original answers	6
Detail animal answers	5	Poorly seen original answers	1
Human form answers	2	Common answers	3
Human form detail answers	10	Percentage of well seen form answers	89
Geographic (map) answers	4	Percentage of well seen animal answers	31
Anatomic answers	3	Percentage of original answers	24
Object	1	Percentage of common answers	10

If one compares the protocols obtained with the subject under the influence of mescaline and when he was in the normal state, one finds remarkable differences in the following respects: 1. The relationship between the whole answers and the small detail answers. In the normal state 35 small detail answers and only 3 whole answers were given; during the mescaline intoxication 8 whole answers and only 6 small detail answers were obtained. (This contrast remains if the longer protocol is abbreviated by taking only the first three answers to each picture.) The protocol taken under the influence of mescaline shows more whole answers than one would expect from the average. On the other hand, the protocol taken in the normal state shows a pronounced small detail type, to such a degree as is only rarely encountered.

As examples of the kind of small detail answers that occurred again and again in the protocol taken while the subject was in the normal state, some answers to picture III may be mentioned (fig. 3). The answer "profile of a man" refers to the place on the blot-picture marked *1*; "profile of a feeble-minded old woman with her hair done high" refers to the small white place marked by the figure *2*, if the picture is held sideways; the "profile of an old man" was seen in the place marked *3*, the picture then being held upside down. In this same picture III, while in the normal state the subject gave 15 small detail answers similar to the ones indicated and no whole answer, he gave, under the influence of mescaline, only one answer, which was: "Two men apparently around a punch bowl, with white aprons and tuxedos on. They have funny faces." (Compare with fig. 3.) This resembles the answer given usually by subjects with good intelligence when shown this picture.

2. When the subject was in the normal state there were many more human detail and animal detail answers than human and animal answers; under mescaline the opposite occurred.

3. In the normal state there were many white answers (9); under mescaline there was only 1.

4. In the normal state there were 4 answers giving only one part of the body alone, when usually the whole body is given as an answer. Under mescaline there were no such answers.

5. In the normal state there were a considerably higher percentage of original answers and a lower percentage of animal answers than under mescaline.

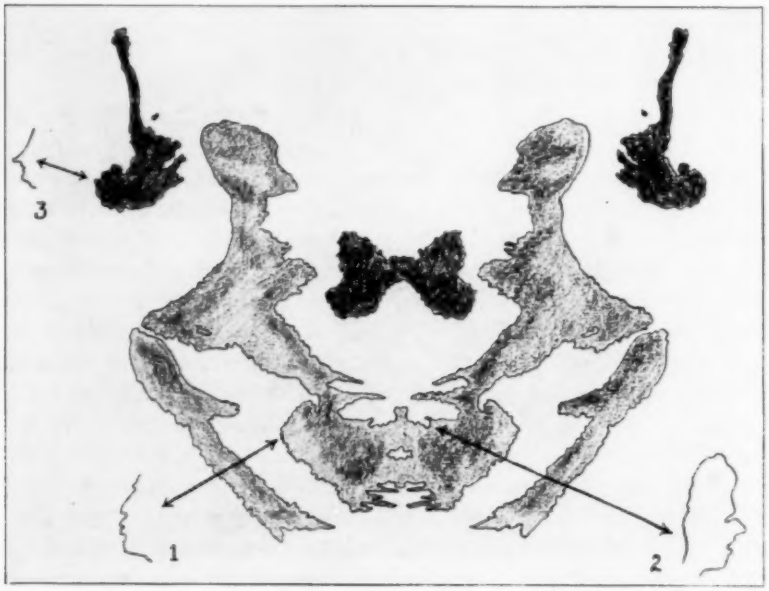

Fig. 3.—Sketch of blot-picture III of the Rorschach test. Examples of the small detail answers given. The small profile (3) was seen in the place indicated when the picture was held upside down.

6. In the normal state there were absolutely and relatively fewer common answers than under mescaline.

These differences, which in part would indicate a diametrical contrast between two personalities, are striking. For the sake of completeness, some similarities between the two protocols may also be added: 1. There is a relationship between dynamic and color answers (3:1.5 and 5:1). 2. In both, there is a good percentage of well seen form answers (89 per cent and 94 per cent) with almost complete absence of poorly seen, original answers (only 1 and 2). 3. Among color answers, only form-color answers are represented; all others are lacking. 4. The

content of the answers is similar; for example, the mentioning of geography and the absence of abstract answers.

In both cases good intelligence, predominantly introverted affectivity, and a certain depressiveness and sociability were mentioned in the interpretation, but with unequal emphasis.

Based on the differences given, mainly on the remarkable contrast in the occurrence of small detail answers in the two tests, the two very different characterizations of personality were reached: that of the analytic, introverted natural scientist, observer and believer in minute details to the extent of missing a grasp of the whole, hesitant and self critical, versus that of the synthetic "intellectual" with a grasp of whole situations, unconcerned with little details, naturally sociable and enjoying the artistic side of life. Even if one feels that the subjective element in making these interpretations is too large a factor, one cannot overlook the difference in significant answers that can be numerically expressed. That this difference has importance for the formulation of the personality structure has been shown by hundreds of tests done by Rorschach himself and by later investigators.

An interesting problem now arises: Which of the two formulations of the personality corresponds to reality, the one given from the protocol obtained under ordinary circumstances or the one obtained when the subject was under the influence of mescaline?

We had assumed *a priori* that the test given under normal conditions would yield within the limits of the test a correct formulation of the personality, and that if there were any differences between the two tests the one given under the influence of mescaline would show the deviations. We were, therefore, surprised to recognize in the test obtained during the intoxication the real personality of the subject as known in life and from his personality study. The characterization deduced from the normal test, on the other hand, although it fitted in some points, described in all important qualities exactly the opposite condition: the subject's distrust of his own originality and of his own intellectual work, exaggerated self-critique, anxiousness and hesitancy in thinking, lack of naturalness and of free emotional life and unrestraint, extremely severe self-control in his thinking and too rigid conscious self-control. All these are qualities that the subject as he was and lived definitely did not show.

A brief abstract of the relevant points of the personality study will show the striking similarity with the interpretation of the data obtained under mescaline intoxication.

The subject of the test was an unmarried economist in the middle twenties of life. It is not necessary here to go into the more concrete data of his life experiences which were obtained during a study of his personality. For the purpose of comparison with the Rorschach test diagnosis it is sufficient to emphasize those categories that are brought out most by this test.

Socially the subject was extraordinarily well adapted. He had many acquaintances with whom he freely discussed his work plans, topics of interest, etc. His social behavior in general was easy and natural. This was noticed especially because he had many rather specialized bookish interests which one might have expected would lead him to a more seclusive "intellectual" attitude. He liked other people and enjoyed being with them.

With his friends he showed great unselfishness, but not of the type that might lead him to forget his own interests entirely. This balance between a healthy self-interest and a lovable unselfishness was paralleled by his intellectual interests. He had broad general cultural interests, covering a number of subjects outside of his own field. These general interests occupied a large part of his time and gave his friends the feeling that he was actually scattering his interests. But just as his unselfishness did not carry him too far, so his general interests never prevented him from following with determination and persistence the course of his material progress and career.

Intellectually, he had creative abilities that had not yet come to any really commensurate fruition, which seemed partly accounted for by the fact that owing to a precocious intellectual development he had perhaps not yet found the real direction of his fundamental interests. His judgment was independent in many respects. His type of intellect was synthetic. He would see broad combinations and connections, and become interested in the whole life work of an author rather than in one of his works. When he had to report on a difficult subject with which he was not familiar he would more or less intuitively select a few books from the library and, without being bothered by difficult details or by conflicting views expressed in the literature, would form his own point of view about the whole field. The result would be a production of considerable merit, good in literary form and sound in content, but perhaps somewhat below the standard that an observer would set for his type of intelligence.

He had no real interest in abstract things, although he occasionally read books on abstract subjects and liked discussing them with his friends. He was not given to close analysis of little details, but he admired the type of scientific work in which this was necessary, and felt that he needed such work as part of his self-education. An example of his attitude in this direction was that after having read an interesting book on the physiology of plants he bought himself a little pocket microscope and spent a Sunday in the fields examining specimens with great interest, but did not repeat the experience.

His disposition was calm and even, with little tendency to untoward emotions. He was very self-controlled. Once, during a difficult conference, when for the first time he had to defend a cause of considerable importance to him, he appeared entirely calm and unconcerned, and won his point by his poise and the friendly, natural presentation of his views. But later he said that he had felt very much upset during the time, to the point of being really sick from excitement.

He was exceedingly well adjusted to his personal and social environment and to the world of affairs. Nevertheless, there was on close observation some unresolved dissatisfaction. Without being able to specify in which field this dissatisfaction lay, the observer could notice that his inner personality, so to say, led an existence of its own which even his closest friends could barely divine beneath his general frankness.

The similarity of these data to the deductions obtained from the Rorschach test given under mescaline are impressive. One may therefore state that the Rorschach test under ordinary circumstances gave a

result that was not representative. During the intoxication the subject reacted very differently, and the result was a formulation of the personality that corresponded closely to the record of the personality description.

It does not seem possible to restrict one's interest to the "correct" protocol, and simply discard the protocol obtained under normal circumstances as wrong. Nor is it a solution simply to discard the Rorschach test as a diagnostic criterion. It is not intended here to go in detail into the possible psychologic explanations of this divergence. They would be difficult to deduce and practically impossible to prove. But the fact of the divergence is emphasized: Under the influence of the intoxication the personality of the subject was clearly indicated by the Rorschach protocol; the characterization from the protocol obtained under normal circumstances was contradictory in many points.

The question arises whether the change in the reaction to the Rorschach test may have been due to a direct influence of the drug, as the hallucinations were, for example, or whether the displacement of the result of the test was a psychologic consequence of a change in attitude. It seems that the result must be due to secondary factors rather than to a direct influence of the poison, for the same doses of poison in other persons caused no such change of results in the same test. There was also in the Rorschach test itself no evidence of hallucinations or pseudohallucinations that would give an indication of the direct effect of the poison. Fatigue phenomena do not seem to have played a part, since it was the protocol taken under mescaline that was the more "normal" one (i. e., came closer to an average protocol).

The personality characterizations given in the discussions cited refer only to the form or kind of reaction, and not to the content of the personality. In recent years one has become habituated in psychiatry to regarding the form of the personality in a large measure as something unaffected by extraneous influences, as something constant. Correlations between physique and personality have tended particularly to lead to such a view. It was the Rorschach test especially that showed that the form of a personality remains the same under the most different circumstances. We also found in our other subjects that the Rorschach protocols under ordinary circumstances and under mescal were usually almost identical.

From the large literature on the Rorschach test, there is no longer any doubt that in the hands of a competent interpreter it reflects correctly to a large extent the formal personality, even though occasional wrong conclusions may naturally occur. The concrete actions in life are answers to stimuli, just as, after all, the answers are in the Rorschach test. One may say, therefore, that our observations give an indication of how much the form of a personality is under the influence of outer circumstances. They further show that the usual psychologic "type"

of a person need not be his only characteristic, but that under the surface other modes of reaction may be hidden. This may be especially true of personalities that appear to be especially clear types. Their personality patterns need not be constant, but may frequently be masks behind which other (contrasting) components of the personality may be hidden. If we find such a contrast in the content and strivings of the psyche, we speak of a repression and a conflict which we have learned to consider as of the greatest importance in psychopathology. The contrast in form and in the mode of reaction that this mescaline experiment has revealed indicates that behind a typical personality pattern there may be a conflicting set of reaction modes which need not, or perhaps cannot, be expressed in terms of content.

SUMMARY

Rorschach tests done on the same subject under the influence of mescaline and under normal circumstances showed a remarkable difference. Of the two Rorschach tests, the one obtained under mescaline, contrary to expectation, corresponded closely to the personality as known in life. The test obtained in normal circumstances, while agreeing in some points, gave a diametrically opposed picture of all important qualities. It is suggested that these observations indicate that the form of a personality is not a constant, but that it also may be influenced by outer circumstances, and that the usual psychologic "type" of a person does not necessarily exhaust the description of the formal structure of his personality.

STUDIES IN SENSATION

III. CHEMICAL FACTOR IN THE STIMULATION OF END-ORGAN GIVING TEMPERATURE SENSATIONS

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Ebbecke [1] drew attention to the temperature sensations that are induced in a limb on release of a circulatory stasis which has been maintained for some while, and subjected these sensations, and the variations in them produced by altering the external conditions, to very careful analysis. He concluded that sensations of both warmth and cold depended on thermal gradients of varying intensity set up at different depths as warm blood entered cooled skin or vice versa. Goldscheider and Hahn [2] subjected Ebbecke's theories to considerable criticism; they showed that if his experiments were repeated quite different sensations, or no sensation at all, might be induced if the time intervals employed were varied. The present experiments were therefore undertaken to measure the actual temperature changes and gradients that accompanied such sensations with a view to applying a more direct test to Ebbecke's theory. In the course of these experiments it became evident that intense sensation of warmth might be induced on release of stasis without any change in temperature level, or the induction of any thermal gradient, so that it would appear logical to assume a chemical stimulus of some type.

METHODS

Stasis was produced by a Riva-Rocci armlet, which compressed the arm above the elbow; in a few experiments the elbow joint was included in the area compressed, so as to exclude collateral circulation through the bone, but this variation of the procedure produced no definite modification of the results. In other experiments the circulation to one finger only was obstructed; in these a small Riva-Rocci type of bag was made from a finger stall, wrapped round the base of the finger and maintained in position by an adhesive plaster bandage. Compression

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This is the last of a series of three papers on sensation: I. Depth, Distribution and Probable Identification in the Prepuce of Sensory End-Organs Concerned in Sensations of Temperature and Touch; Thermometric Conductivity, Arch. Neurol. & Psychiat. **27**:489 (March) 1932; II. The Mode of Stimulation of Cutaneous Sensations of Cold and Warmth, ibid. **27**:1031 (May) 1932.

1. Ebbecke: Arch. f. d. ges Physiol. **169**:395, 1917.

2. Goldscheider and Hahn: Arch. f. d. ges Physiol. **206**:337, 1924; **208**:559, 1925.

was produced rapidly; a large glass reservoir was used, in which a considerable pressure could be induced, and this was then put suddenly into connection with the armlet. During compression, the pressure was maintained at about 200 mm. of mercury (extreme limits 180 and 220 mm.).

Temperature measurements were made with the smallest McGlone thermocouple loops of constantan and manganin, already described by us,[3] connected to D'Arsonval galvanometers of three second periods. The galvanometer excursions were recorded photographically (1 meter distance). Three thermocouples were usually employed, one situated on the surface and the others at depths of 1 or 2 mm. The depth was determined by x-ray photographs. The surface temperature was determined by comparison with a junction maintained at a constant temperature in a thermos flask within a thermoregulated jar, as previously described by us;[4] in most experiments the sensitivity employed was about 1 cm. per degree centigrade, allowing the temperature level to be read with an accuracy of about ± 0.05 C. and changes in temperature with one of ± 0.02 C., but when the range of temperatures employed was very great this sensitivity had to be reduced to 3 or 4 mm. per degree centigrade, with a consequent loss in accuracy. In the majority of the experiments the forearm was immersed to the elbow joint in a water bath maintained by a thermoregulator at a definite temperature and vigorously and continually stirred. The water temperature was recorded with a resistance thermometer (Cambridge pattern). Even with the lower sensitivity, rarely did the two estimations differ by more than 0.1 C. The thermoregulator maintained a temperature constant (± 0.05 C.) and the bath volume was large, so that fluctuations were slow and did not give rise to sensations.

The buried couples in all the experiments in water were of the differential type previously described by us;[3] they consisted of two loops, one buried in the tissues and the other strapped to the surface. The constantan had an unbroken circuit between the two loops, and the two manganin wires were soldered to copper, and both copper-manganin junctions were insulated and enclosed in a single sheath of copper foil, so that their temperatures were identical. The surface thermocouples of these differential couples were placed as close as possible to that recording surface temperature (within a few millimeters). In this way spatial gradients were recorded directly, and temperature levels at a depth indirectly by comparison with the other surface thermocouple. The sensitivity employed for the differential couples was 1.2 cm. per degree centigrade so that changes in gradient could be determined with considerable accuracy.

In a few experiments stasis was produced and released with the forearm in air at ordinary room temperature; in most it was immersed in the water bath already described. An interval of twenty minutes or more was usually allowed for the tissues to attain thermal equilibrium before experimental procedures were commenced. Attempts have also been made to repeat Ebbecke's experiment, where the forearm is kept in water during stasis but is removed and rapidly dried before the stasis is released; in such experiments the thermocouples have to be introduced after drying, since effective drying is impossible with the thermocouples in place. For this reason needle thermocouples (of the type previously described) were employed in some experiments, and were inserted obliquely after stasis had been induced.

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3. Bazett, H. C., and McGlone, B.: Am. J. Physiol. **82**:415, 1927; J. Physiol. **64**:393, 1928.

4. McGlone, B., and Bazett, H. C.: Am. J. Physiol. **82**:452, 1927.

In order to conduct experiments in which the temperature of the inflowing blood on release could be confidently assumed to be identical with that of the tissues into which it flowed, a resistance thermometer was also inserted into the rectum, and continuous records were made on a Gamgee thread recorder of the rectal temperature as well as that of the bath. The resistance thermometers employed were of platinum wire contained in a silver tube (modeled after the Cambridge pattern), so arranged that the proximal end of the silver tube was inserted 13 cm. beyond the anus. The recorder was that made by the Cambridge Instrument Company.

It was found that if the bath was maintained exactly at the same temperature as that of the rectum, on release of stasis reverse gradients were set up and the tissues were slightly cooled by the incoming blood. This was explicable as the result of the cooling of the arterial stream in its course through the upper part of the arm, a cooling described by Bernard [5] and demonstrated by Stewart,[6] which has already been referred to in earlier papers.[7] In order to avoid the production of such gradients, the bath was in other experiments maintained a few tenths of a degree centigrade below the rectal temperature level.

Three subjects, B, F and M, were employed. The sensations experienced by the subject were signaled by a tambour-mirror system on photographic paper. Unless otherwise stated all sensations mentioned were referred by the subject to the area in which the thermocouples were placed; in most cases this was the extensor surface of the forearm or hand.

A few control experiments were made to determine the rate of change of temperature in the bath that could be detected by the subjects with the exposure to the bath of this large area of skin.

STASIS OF THE FOREARM IN AIR

During stasis there is a definite but very gradual cooling of the tissues, and on release of stasis the previous temperature is regained and a still higher level is attained as the result of the reactive hyperemia. The heat capacity of the tissues, however, is so great that the changes are very slow. Illustrative experiments are shown in table 1. It will be noticed that on release of stasis an initial slight fall of temperature might be recorded, then the temperature rose slowly, except when initial temperatures were very low after the arm had been cooled in water, when the changes were much more rapid. The initial fall of temperature on release may have depended on blood entering from a cooler area; a similar fall was often seen in the bath experiments and sometimes a rise was seen during the induction of stasis. Rarely did such changes exceed 0.05 C.; they were more probably an artefact. Induction of stasis caused some congestion of the limb and tended to form a groove in which the surface thermocouple lay, and on release this congestion disappeared. These slight changes may therefore have

5. Bernard, Claude: Leçons sur la chaleur animale, Paris, J. B. Ballière et fils, 1876.

6. Stewart, G. N.: Heart **3**:33 and 88, 1911.

7. Bazett and McGlone (footnotes 3 and 4).

TABLE 1.—*Release of Stasis in Air*

Experiment	Conditions		Resting Level Before or After Stasis	Before Release	Sensation 1	Sensation 2	Sensation 3	Sensation 4	Maximum Temperature Recorded	Comment
1	Subject M: arm in air; stasis 9 min.; loop thermocouples in forearm; room temp.	Time in secs.	.....	0	3	5	7	24	720	Room temp., dry bulb, 26.2; wet bulb, 22.3 C.
		Sensation	.....	.....	(Hand warm)	(Hand cold)	Cold	Still cold	.....	
		Temp. at 0.65 mm	34.27	33.35	33.35	33.35	33.35	33.46	34.32	
		Temp. at 2.6 mm	34.61	33.66	33.66	33.66	33.66	33.76	34.69	
2	Subject M: arm in air bandaged before stasis; stasis 27½ min.; needle thermocouples in forearm	Time in secs.	.....	.....	2	6	11	19	180	Room temp., dry bulb, 25 C.; wet bulb, 13.7 C.
		Sensation	.....	.....	Warm	Cold	Warm surge	Cool	Tingling	
		Temp. at surface	.....	32.00	31.98	32.05	32.06	32.08	33.95	
		Temp. at 1.8 mm	34.15	32.16	32.00	32.26	32.30	32.42	34.90	
3	Subject B: arm in water at 17 C. for 10 min.; stasis induced and maintained 14 min. while arm dried and needle thermocouple inserted in hand; room temp.	Time in secs.	.....	0	6	15	36	51	240	Room temp., dry bulb, 24.7 C.; wet bulb, 13.7 C.
		Sensation	.....	.....	(Intense warmth in arm)	Warmth	Burning	Tingling	Tingling	
		Temp. at surface	.....	22.30	22.14	22.30	23.47	24.64	25.48	
		Temp. at 2.1 mm	35.24	26.68	26.60	27.31	28.72	29.68	30.31	
4 (a)	Subject M: arm in water at 15.7 C. for 9 min.; stasis in water 1 min. in air with partial drying 4 min.; loop couples in hand; rectal temp., 37.09 C.	Time in secs.	.....	0	5	17	31	78	60 min.	Room temp., dry bulb, 24 C.; arm and hand very red, no cyanosis; hyperemic response on release indefinite; sensation mild but definite
		Sensation	Cold	.....	Warm	Cool	Still cool	Slight tingling	.....	
		Temp. at surface	16.27	21.68	21.68	21.74	21.94	22.60	29.65	
		Temp. at 0.85 mm	17.68	21.85	21.87	21.89	22.09	22.63	29.83	
(b)	Arm in water at 15.7 C. for 9 min.; stasis in water 1 min. in air with partial drying 15 min.; rectal temp., 39.6 C.	Time in secs.	.....	0	3	6	30	76	50 min.	Cyanosis and Bier's spots after stasis for 10 min.; cyanosis disappearing and hyperemia in evidence 15 sec. after release; cool sensations on back of hand slight
		Sensation	Cool	.....	Warm	Slightly cool	Neutral	Tingling ++	.....	
		Temp. at surface	16.14	21.05	21.06	21.05	21.21	22.09	23.77	
		Temp. at 0.85 mm	17.01	20.98	21.00	20.99	21.13	22.06	23.70	
(c)	Arm in water 15.7 C. for 11 min.; removed to air and partially dried without stasis (as control)	Time in secs.	.....	.....	2	8	35	100	170	Hand moderately dry 15 secs. after removal from water
		Sensation	.....	.....	Warm	Warmer	Warm	Warmer	Slightly cool	
		Temp. at surface	16.16	.....	16.13	16.74	17.77	18.98	19.71	
		Temp. at 0.85 mm	16.47	.....	16.59	16.72	17.75	18.96	19.70	
(d)	Arm in water at 15.65 C. for 27 min.; stasis for 15 min. and release in water for comparison; rectal temp., 37.25 C.	Time in secs.	.....	0	2	27	65	.....	101	Slight cyanosis at end of stasis and slight hyperemia about 27 secs. after release; warm sensation developed after stasis for 10 min. and little altered on release; temp. sensations on release all slight
		Sensation	.....	Warm	Warm	Slightly cool	Tingling	.....	Tingling ++	
		Temp. at surface	15.85	15.78	15.78	15.78	15.84	.....	15.88	
		Temp. at 0.85 mm	16.00	15.79	15.80	15.80	15.86	.....	15.90	

resulted from slight variations in the effective depth of the surface thermocouples. The sensations were more intense when the temperature changes and temperature gradients were greater, but they appeared to be induced when no measurable change in temperature had occurred and might be quite intense even when the temperature changes were slow. In experiment 2, the sensations appeared to occur in surges, and waves in the rise of temperature were particularly in evidence; a rise of temperature of less than 0.1 C. might apparently give rise to cold sensations, followed later, when the rate of change was much less, by warmth. However, no constant relationship of this type was observable either in this or other experiments, nor any to the measurable distribution of spatial gradients.

The complexities that arise with experiments of Ebbecke's original type are illustrated in experiment 4. The sensations of cold were rather less marked with stasis for fifteen minutes instead of five minutes, but repetition of the experiment introduced another variable factor in the gradual progressive cooling of the whole forearm, even though intervals of from fifty to sixty minutes in air were allowed for recovery. The cooling of the deeper tissues lowered the temperature of the arterial supply to the skin, so that on each successive immersion the temperature, at a depth of 0.85 mm., fell nearer to that of the water, and the temperature reached on recovery was lower. The original temperatures before immersion in water were: at the surface, 32.85 C.; at a depth of 0.85 mm., 32.91 C. The slight reversed gradients seen in recovery in (*b*) and (*c*) may have depended to a nearness of the deeper thermocouple to some venous channel coming from a more peripheral area, but they, and the warm sensation experienced in (*c*), may have resulted from the cooling of the tissues to a level considerably below the air temperatures. This level may even have been near or below the wet-bulb temperature, since the arm was held close above the large bath of rapidly circulating water, and the humidity must have been much above that of the rest of the room.

STASIS OF THE FOREARM IN WATER

When the forearm was immersed in water at about body temperature, release of stasis produced no appreciable thermal change or thermal gradient, yet intense sensations of warmth were consistently induced. It was impossible to adjust the temperature of the bath at this temperature level so as to prevent the occurrence of these sensations. An example of such an experiment is shown in figure 1, and an abbreviated protocol of this experiment (experiment 5) is also given. Further instances of similar results may be seen in table 2, experiment 6 (*e*) and (*f*), and experiment 7 (*a*), (*b*) and (*c*).

A SAMPLE PROTOCOL

EXPERIMENT 5 (subject B).—The room temperature was 26.8 C. dry bulb. Thermocouples were placed on the back of the hand at depths of 0.6 and 1 mm. In air the temperatures were: surface, 34.98 C.; at 0.6 mm., 35.43 C.; at 1 mm., 35.51 C.; rectal, 36.90 C. The hand and forearm were immersed in a bath at from 37.13 to 37.16 C. for thirty minutes, when stasis was caused by compression of the upper part of the arm for five minutes. Before induction of stasis, there was still a slight sensation of warmth; after stasis for one minute this was increased and was later accompanied by some tingling. Part of the record

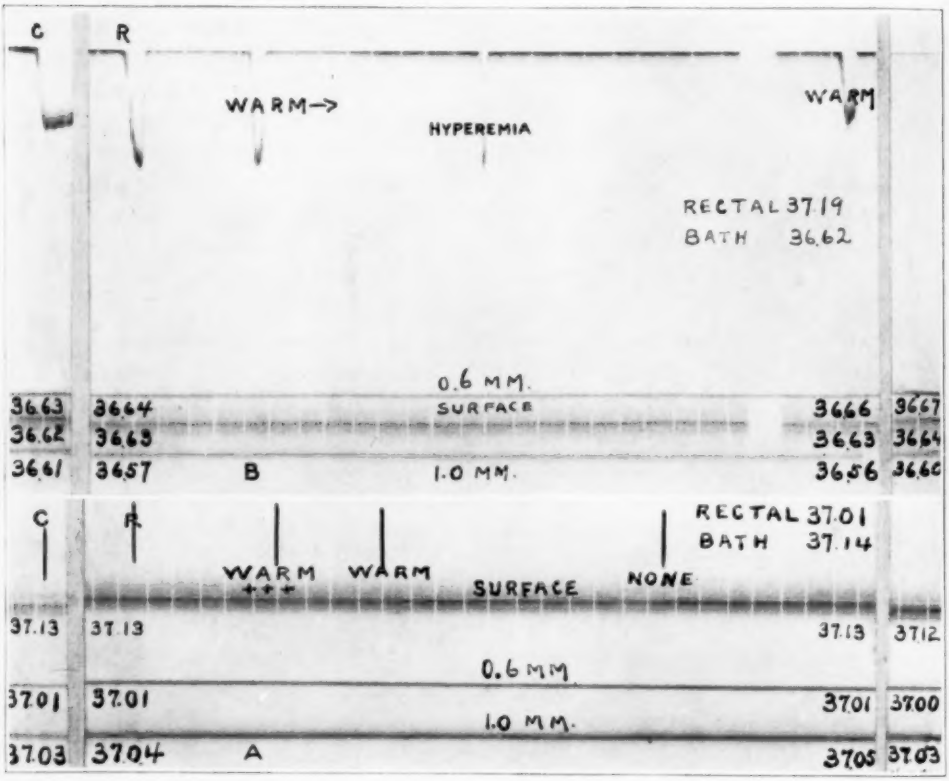

Fig. 1.—*A*, records obtained on release of stasis of the forearm for 5 minutes in a bath at 37.14 C. (see experiment 5). The three records are as labeled; the signal indicating sensation is not reproduced (to save space) but the signals are indicated. *C* indicates the moment of compression; *R*, the moment of release. The final portion of the record indicates temperatures 160 seconds after release. The time marker indicates periods of 1.02 seconds. *B*, records obtained on release of stasis for 5 minutes in a bath at 36.62 C. Signals are shown as recorded. The final portion of the record indicates temperatures 80 seconds after release.

obtained on release is shown in figure 1*A*; the sensations were as there indicated; after thirty-two seconds., "slightly cool or neutral;" after forty-two seconds, "mild tingling." After a rest of seventeen minutes, stasis was repeated and con-

tinued for ten minutes. During stasis, a sensation of warmth developed after two minutes, followed by slight tingling, and a sensation "almost hot" after four minutes. On release, the sensations were: six seconds, "very warm or hot"; twenty-four seconds, "sudden cessation of warmth? cool"; fifty-four seconds, tingling becoming intense and lasting a considerable time. The temperature sensations were only slightly more intense than on previous release. The temperatures were: bath, 37.18 C.; surface, 37.11 to 37.14 C.; at 0.6 mm., 37.03 to 37.08 C.; at 1 mm., 37.10 to 37 C. throughout the period of stasis and release. Cyanosis during stasis was definite, but the hyperemia response on release was not noticed for seventeen seconds. The hyperemia lowered the temperatures slightly; after fifty-four seconds, when the change was maximal, the falls were 0.03 C. at the surface, 0.06 C. at 0.6 mm. and 0.03 C. at 1 mm.

Since the thermal gradients were reversed, the bath temperature was lowered to 36.64 C. and after twenty minutes stasis was applied for ten minutes. A sensation of "slight cool" changed during stasis through "slight warmth" "definite warmth" "almost hot" to "moderate warmth and numbness"; the arm was cyanosed. On

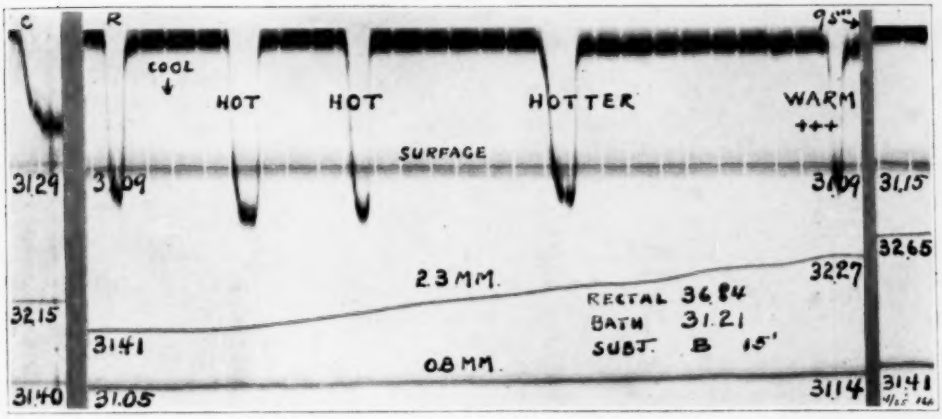

Fig. 2.—Part of the record obtained in the experiment detailed in table 2 (experiment 6*d*) on release of stasis of the forearm for 15 minutes in a bath at 31.21 C. The final record indicates temperatures 130 seconds after release.

release, the sensations were: five seconds, warm; nine seconds, very hot; twenty-eight seconds? slightly cool; fifty-three seconds, tingling. Hyperemia was definite after eighteen seconds. The temperatures were: before stasis, 36.64 C. at all depths; rectal, 37.14 C.; during stasis the temperature at 1 mm. appeared to fall to 36.60 C., and during reactive hyperemia the temperatures rose to a maximum after ninety seconds: of the surface, 36.66 C.; at 0.6 mm., 36.72 C. and at 1 mm. 36.66 C. After a rest of twenty minutes, stasis was repeated for five minutes and part of the record is shown in figure 1*B*. The sensations were less intense. After thirteen minutes' rest, stasis was repeated for two and one-half minutes. The sensations on release were similar, but still less intense, and there was practically no tingling.

Comment.—Experiments 5, 6 and 7 amply demonstrate that intense sensations of warmth may be induced by release of stasis even when temperature changes of less than 0.05 C. are involved, and when such

changes do not exceed 0.02 C. per second; the sensations of warmth are independent of the direction of change in temperature. They are produced in a similar manner whether a reverse gradient of about 0.2 C. per millimeter is present, or a positive gradient of about 0.06 C. per millimeter, and it is immaterial whether the tissues have a temperature very slightly above or below that of the entering blood.

The examples given in table 2 and those reproduced in figures 2 and 3 demonstrate the variations in sensation induced by varying the temperature of the bath or duration of stasis. During stasis at the lower temperature levels, an intensification of cool sensation resulted immediately but decreased gradually during the period of stasis; at the higher temperature levels there was no immediate sensation, but after a few minutes (the time being shorter the higher the temperature)

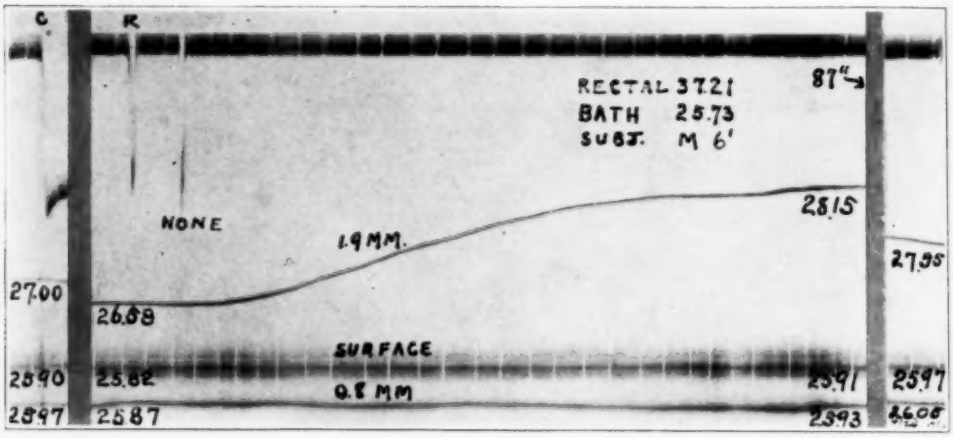

Fig. 3.—Part of the record obtained in the experiment detailed in table 2 (experiment 7*e*) on release of stasis for 6 minutes in a bath at 25.73 C. The final record indicates temperatures 119 seconds after release.

a sensation of warmth gradually developed, often became intense and the sensation was often called "almost hot," though the characteristic hot never appeared to be really definite. These sensations of warmth usually partly faded or were replaced with sensations of numbness or tingling near the close of the longer periods of stasis. On release of stasis at the lower temperature levels, sensations of cold predominated in subject B (experiment 6), but were less marked with long than with short periods of stasis. On release at such temperatures, a cool sensation experienced at the end of stasis might be increased on release (experiment 6 *a*, with stasis for five minutes) or might be decreased (experiment 6 *b*, with stasis for ten minutes). In subject M, at these low temperatures the sensations on release were predominately warm,

after stasis for ten minutes. At temperatures about 30 C., sensations fluctuating between warmth and cold were common (experiment 6 *c* and *d*) and with the longer periods of stasis the warm sensations were more marked. At the higher temperature, sensations of warmth only were experienced, and were usually more intense the longer the period of stasis. At high and moderate temperature, sensations called "hot" were common on release.

At certain temperature levels, release of stasis might produce little or no definite sensations of temperature according to the duration of the stasis. In subject M, at 25.75 C., stasis for six minutes gave such a result, while stasis for ten minutes at the same temperature produced only slight warmth (experiment 7 *e* and *f*). Stasis for fifteen minutes at 15.6 C. (experiment 4 *d*) similarly gave little sensation of temperature in this subject. In subject F, a similar absence of definite sensations resulted from release of a five minute stasis at 23.3 C.

The presence of surges in the temperature changes was common on release of stasis (figs. 2 and 3).

COMPARISON OF STASIS OF ONE FINGER ONLY WITH THAT OF WHOLE FOREARM

The sensations induced by the presence and release of stasis usually were most marked in the hand, but were also referred to the fingers and forearm. When, however, the circulation of one finger only was obstructed, the sensations experienced in that finger were quite different. Some examples are given in table 3. It may be noted that at moderate temperatures, with the finger only involved in stasis, the sensations were predominately cold and with stasis of the whole arm, predominately warm, yet the temperature changes were not greatly different. At the higher temperatures, warmth only was induced by release of stasis to the whole arm, but cool was also noted when the finger only was involved. At intermediate temperatures the same exaggeration of cool sensations and minimizing of warm sensations was noted when the arm was not involved in the stasis (experiments 6 and 9). The sensations were much the same, when stasis to the finger only was employed, whether the whole forearm or the finger only was immersed in the bath (experiment 9). Among the temperatures quoted in experiment 8 are many in which the temperatures recorded within the finger appeared to be below even that of the bath; these apparent low values were probably due to the presence of some undetected error (e. g., a parasitic current not present during standardization). No emphasis should be placed on these actual levels. The experiment quoted indicates the maximum values of such discrepancies which were rarely so great.

TABLE 2.—*Release of Stasis of Varying Duration in Water at Varying Temperature*

Experiment	Conditions		Before Stasis	Before Release	Sensation 1	Sensation 2	Sensation 3	Sensation 4	Maximum Change Recorded	Comment
6 (a)	Subject B; forearm in bath at 12.75 C. for 63 min. then stasis 5 min.; couples in forearm; rectal temp., 36.9 C.	Time in secs.	.....	0	5	8	.....	.....	49	Arm red, no cyanosis or reactive hyperemia noted
		Sensation	Cool	Slightly cool	Tingling	Slightly cool	.....	.....	.....	Tingling very slight
		Temp. at surface	12.78	12.88	12.74	12.77	.....	.....	12.74	Sensation of cold greatest at commencement of stasis
		Temp. at 0.8 mm	13.00	13.13	13.01	13.06	.....	.....	13.01	
		Temp. at 2.3 mm	14.12	14.02	14.01	14.09	.....	.....	15.16	
	(b) As in (a) after rest of 10 min.; stasis for 15 min.	Time in secs.	.....	0	11	31	.....	.....	75	Arm slightly purplish red after 10 min., stasis returning to bright red soon after release; slight sensation of cold at commencement of stasis
		Sensation	.....	Cool	Slightly cool	Tingling	.....	.....	Tingling	
		Temp. at surface	12.67	12.75	12.75	12.75	.....	.....	12.72	
		Temp. at 0.8 mm	12.91	12.91	12.93	12.91	.....	.....	12.92	
		Temp. at 2.3 mm	14.32	13.59	13.93	15.40	.....	.....	16.02	
	(c) Bath at 31.2 C. for 45 min., then stasis for 5 min.; rectal temp., 36.8 C.	Time in secs.	.....	0	1	3	5	18	53	No definite cyanosis; slight hyperemia flush 3 secs. after release; tingling very slight
		Sensation	.....	Slight tingling	Warm	Cool	Warm	Warm, tingling	Slightly warm	
		Temp. at surface	31.20	31.17	31.17	31.17	31.20	31.20	31.26	
		Temp. at 0.8 mm	31.22	31.19	31.19	31.21	31.24	31.22	31.34	
		Temp. at 2.3 mm	31.96	31.63	31.67	31.67	31.66	31.88	32.20	
	(d) As in (c) after rest of 16 min.; stasis for 15 min.	Time in secs.	.....	0	3	5	17	31	93	Cyanosis after 6 min.; marked hyperemia about 5 secs. after release; see figure 2
		Sensation	.....	Warm?	Slightly cool	Very hot	Hotter	Very warm	Tingling ++	
		Temp. at surface	31.29	31.09	31.09	31.09	31.09	31.09	31.09	
		Temp. at 0.8 mm	31.40	31.05	31.05	31.08	31.12	31.14	31.43	
		Temp. at 2.3 mm	32.15	31.41	31.41	31.40	31.93	32.37	32.89	
	(e) Bath at 37 C. for 35 min., then stasis for 5 min.; rectal temp., 37 C.	Time in secs.	.....	0	6	16	35	55	.....	Marked cyanosis during stasis; hyperemia 6 secs. after release; marked sensation of warmth developing during stasis
		Sensation	Warm	Very warm	Less warm	Warm	Very warm	Slightly warm	.....	
		Temp. at surface	37.08	37.05	37.04	37.04	37.03	37.05	.....	
		Temp. at 0.8 mm	36.82	36.90	36.90	36.90	36.89	36.85	.....	
		Temp. at 2.3 mm	36.89	36.79	36.80	36.78	36.79	36.78	.....	
	(f) As in (e) after rest of 15 min.; stasis for 15 min.; bath falling to 36.95 C.; rectal temperature, 37.09 C.	Time in secs.	.....	0	5	8	19	22	.....	Marked cyanosis developed within 6 min. and reactive hyperemia 3 secs. after release; marked sensation of warmth during stasis after 4 or 5 min., becoming numb ache at end
		Sensation	.....	Ache	Warm	Very hot	Still hot	Tingling	.....	
		Temp. at surface	37.04	36.97	36.98	36.98	36.98	36.98	.....	
		Temp. at 0.8 mm	36.75	36.70	36.71	36.71	36.71	36.71	.....	
		Temp. at 2.3 mm	36.90	36.88	36.88	36.88	36.88	36.86	.....	

(g) Bath at 40.3 C. for 30 min., then stasis for 5 min.; rectal temp., 37.20 C.	Time in secs.....	.....	0	4	10	21	75	118	Cyanosis definite within 2 min., hyperemia on release within 4 secs.; sensation of marked warmth or hot during stasis developing within 1 min. and decreasing before release
	Sensation.....	Warm	Warm	Less warm	Very warm	Less warm	Neutral	Slightly warm	
	Temp. at surface.....	40.33	40.34	40.33	40.33	40.36	40.33	40.33	
	Temp. at 0.8 mm.....	39.77	39.90	39.90	39.87	39.86	39.62	39.63	
	Temp. at 2.3 mm.....	39.00	39.63	36.64	39.63	39.47	39.18	38.94	
7 (a) Subject M; forearm in bath at 37 C. for 17 min., then stasis for 5 min.; couples in back of hand; rectal temp., 37.25 C.	Time in secs.....	.....	0	2	8	33	.....	.....	Hyperemia on release but time not noted
	Sensation.....	.....	Tingling	Hot	Very warm	Neutral	.....	.....	
	Temp. at surface.....	37.29	37.30	37.30	37.30	37.30	.....	.....	
	Temp. at 0.8 mm.....	37.21	37.22	37.22	37.22	37.23	.....	.....	
	Temp. at 1.9 mm.....	37.02	37.14	37.14	37.14	37.16	.....	.....	
(b) As in (a) after rest of 15 min.; stasis for 10 min.; rectal temp., 37.35 C.	Time in secs.....	.....	0	3	6	13	24	.....	Cyanosis after 6 min.; cutenea marmorator near close; hyperemia within 6 seconds on release; water felt very warm, almost hot toward end of stasis; sensations on release of similar or less intensity than in (a)
	Sensation.....	.....	Very warm	Warmer	Hot	Tingling	Cool	.....	
	Temp. at surface.....	37.28	37.28	37.28	37.28	37.28	37.28	.....	
	Temp. at 0.8 mm.....	37.20	37.25	37.25	37.25	27.27	37.27	.....	
	Temp. at 1.9 mm.....	37.16	37.16	37.16	37.17	37.17	37.18	.....	
(c) After 30 min. in bath at 36.5 C. stasis for 5 min.; rectal temp., 37.37 C.	Time in secs.....	.....	0	1.3	19	39	72	.....	Cyanosis not definite—hyperemia slight, indefinite and hard to time
	Sensation.....	.....	.....	Hot	Very warm	Warm	Neutral	.....	
	Temp. at surface.....	36.57	36.55	36.55	36.55	36.56	36.56	.....	
	Temp. at 0.8 mm.....	36.49	36.47	36.47	36.47	36.51	36.52	.....	
	Temp. at 1.9 mm.....	36.54	36.50	36.51	36.68	36.70	36.69	.....	
(d) After 30 min. in bath at 39.85 C. stasis for 5 min.; rectal temp. 37.31 C.	Time in secs.....	.....	0	1.5	3	19	68	80	Cyanosis probably present but neither cyanosis nor onset of hyperemia noted; sensations similar in intensity to those of previous releases
	Sensation.....	Slightly warm	.....	Warm	Hot	Warm	Slightly cool	Tingling	
	Temp. at surface.....	39.79	39.87	39.86	39.88	39.86	39.81	39.72	
	Temp. at 0.8 mm.....	39.48	39.63	39.61	39.62	39.60	39.52	39.41	
	Temp. at 1.9 mm.....	38.94	39.46	39.43	39.43	38.87	38.51	38.40	
(e) After 60 min. in bath at 25.75 C. stasis for 6 min.; rectal temp. 37.23 C.	Time in secs.....	.....	0	46	88	.....	.....	32	No cyanosis during stasis; no definite hyperemia on release; see figure 3
	Sensation.....	.....	.....	Tingling	Slightly cool	.....	.....	.....	
	Temp. at surface.....	25.90	25.82	25.82	26.01	.....	.....	25.91	
	Temp. at 0.8 mm.....	25.97	25.87	25.91	26.07	.....	.....	25.93	
	Temp. at 1.9 mm.....	27.00	26.58	28.15	27.84	.....	.....	28.15	
(f) As in (e) after 12 min. rest; stasis for 10 min.	Time in secs.....	.....	0	3	5	14	39	63	Slight cyanosis after 8 min. stasis and sensation of warmth at same time
	Sensation.....	.....	Slightly warm	Slightly warm	Cool	Slightly tingling	Neutral	Tingling	
	Temp. at surface.....	25.82	25.86	25.86	25.90	25.92	25.81	25.89	
	Temp. at 0.8 mm.....	25.84	25.83	25.83	25.85	25.89	25.80	25.94	
	Temp. at 1.9 mm.....	26.65	26.31	26.35	26.42	27.13	27.66	28.04	

TABLE 3.—*Comparison of Stasis of Finger Only With That of Whole Forearm During Immersion in Water* *

Experiment	Conditions		Before Stasis	Before Release	Sensation 1	Sensation 2	Sensation 3	Sensation 4	Maximum Change Recorded	Comment
8 (a)	Subject B: forearm and hand in water at 26.05 C. for 26 min., then stasis of middle finger only for 10 min.; temp. of water fell to 25.95 C. during experiment; couples on extensor surface of middle finger and 1.5 mm. deep to this; rectal temp., 26.86 C.	Time in secs.....	.....	0	1.5	4	6.5	10.5	80	Slight cyanosis after stasis for 6 min.; on release, slight hyperemia in 2 secs. disappearing in 6 secs.; slight sensation of warmth during stasis disappearing before end of stasis; on release, cool sensation maintained for 40 secs.
		Sensation.....	.....	.....	Slightly warm	Cool	Very cool	Mixed	Neutral	
		Temp. at surface....	25.91	25.89	25.87	25.89	25.88	25.89	25.93	
		Temp. at 1.5 mm....	25.99	25.73	25.73	25.74	25.86	25.96	26.59	
	(b) After rest of 34 min,. stasis to whole arm for 10 min.; water temp., 26.1 to 26.2 C.; rectal temp., 36.78 C.	Time in secs.....	....	0	1.5	3	6	10	63	Cyanosis after stasis for 6 min.; on release marked hyperemia; no sensation except tingling during stasis; on release, warm sensation maintained for 55-80 secs.
		Sensation.....	.....	.....	Slightly warm	Slightly cool	Warm	Hot	Tingling	
		Temp. at surface....	25.97	26.08	26.08	26.06	26.05	26.01	26.12	
		Temp. at 1.5 mm....	26.15	25.99	25.99	25.99	25.95	25.92	26.51	
	(c) After rest of 24 min. stasis to finger only for 10 min.; water temp., 26.3 C., rectal temp., 36.71 C.	Time in secs.....	.....	0	1.5	3	6	10	79	Marked cyanosis after 9 min. disappearing 1.5 secs. after release; no sensation of warmth noted during stasis; on release cool sensation maintained for 48 secs.
		Sensation.....	Slightly cool	Slightly cool	Warm	Cool	Very cool	Mixed	Neutral	
		Temp. at surface....	25.14	26.14	26.17	26.18	26.18	26.17	26.20	
		Temp. at 1.5 mm....	26.37	26.05	26.09	26.09	26.11	26.11	27.00	
	(d) After experiments at 31 C. and rest in water at 42.5 C. for 33 min., stasis of finger only for 5½ min.; rectal temp., 37 C.	Time in secs.....	.....	0	2.5	3.5	2.3	.....	52	Cyanosis developing within ½ min. and disappearing 2 or 3 secs. after release; no marked reactive hyperemia; sensation of warmth after stasis for 17 secs., hot after 1½ min., milder at end; on release, warmth for 30 secs.
		Sensation.....	Slightly warm	Slightly hot	Slightly cool	Warm	Slightly warm	.....	Slightly warm	
		Temp. at surface....	42.07	42.32	42.32	42.33	42.15	.....	42.09	
		Temp. at 1.5 mm....	40.14	42.37	42.36	42.38	40.93	.....	40.20	
	(e) After rest of 12 min. stasis to whole arm for 5 min.; bath temp., 42.2 C.; rectal temp., 37.16 C.	Time in secs.....	.....	0	2	5.5	10.5	21.5	116	Stasis giving effects as in (d) but with also sensation of tension; on release hyperemia greater than in (d); sensation rather longer in duration
		Sensation.....	Slightly warm	Hot	Slightly warm	Hot	Warm	Slightly warm	Slightly warm	
		Temp. at surface....	41.86	42.18	42.18	42.18	42.17	42.05	41.59	
		Temp. at 1.5 mm....	39.73	42.09	42.07	42.09	41.95	41.21	39.46	
9 (a)	Subject M: fingers only in bath at 28.6 C. for 100 min., then stasis of middle finger only for 10 min.; couples on extensor surface of finger and 1.8 mm. deep to it	Time in secs.....	.....	0	1.5	4	9	22.5	39.5	During stasis slight cyanosis, and sensation of warmth and tingling diminishing near end of stasis
		Sensation.....	.....	Slightly warm	Warm+	Cool	Cooler	Slightly cool	Neutral	
		Temp. at surface....	29.24	28.57	28.57	28.62	28.68	28.94	29.06	
		Temp. at 1.8 mm....	30.24	28.62	28.62	28.59	28.84	29.52	29.98	
	(b) Hand and forearm in water at 28.6 C. for 18 min., then stasis of finger only for 10 min.	Time in secs.....	.....	0	1.5	3	7.5	19.5	53	During stasis slight sensation of warmth, fading later; on release pt. sensation weaker, 3rd stronger than in (a)
		Sensation.....	.....	.....	Warm	Cool	Cool	Mixed	Slightly warm?	
		Temp. at surface....	29.27	28.62	28.62	28.65	28.70	29.08	29.50	
		Temp. at 1.8 mm....	30.06	28.61	28.60	28.61	28.79	29.58	30.45	
	(c) Hand and forearm in water at 34.35 C. for 24 min.; stasis of finger only for 10 min.; water temp. falling to 34.2 C. during experiment	Time in secs.....	.....	0	1	3.5	4	18	42	During stasis slight sensation of warmth fading later; onset of cold sensation (3) quite abrupt, and lasting some 14 secs.
		Sensation.....	.....	Slightly warm?	Warm	Warmer	Cold	Mixed	Slightly warm?	
		Temp. at surface....	34.39	34.18	34.18	34.19	34.20	34.44	34.68	
		Temp. at 1.8 mm....	34.72	34.20	34.20	34.21	34.22	34.76	35.16	

* Owing to the position of the hand, it was impossible to distinguish accurately the presence or absence of cyanosis or hyperemia in the finger.

SENSATIONS OF TEMPERATURE PRODUCED BY VARIATIONS IN THE BATH TEMPERATURE

It has been stated that fluctuations in the bath temperature gave rise to no sensations of temperature; it seemed, however, important to determine what the threshold stimulus was when such a large area was involved. When the temperature of the bath was changed to a new level, marked sensations of temperature were induced, which only faded gradually. For instance, in experiment 6, subject B, the initial temperatures in air were: surface, 32.69 C.; at 0.8 mm., 32.98 C.; at 2.3 mm., 33.20 C. After immersion in the bath at 12.75 C. for sixteen minutes, the initial pain had disappeared, but cold sensations were still marked; the temperatures were: 12.74 C., 13.45 C. and 15.13 C., respectively; the fall of temperature was at this time very slow in the superficial layers, for ten minutes later the temperatures were 12.76 C.; 13.20 C. and 14.35 C.; yet even after sixty-three minutes a sensation of cool was still present, though for twenty minutes no fall of temperature had been detectable on the surface or at 0.8 mm.; at 2.3 mm., the temperature had fallen 0.1 C. and returned to the earlier level. At this time the spatial gradient in the most superficial layer was slightly greater than the original gradient in air (0.36 C. per millimeter increased to 0.40 C. [experiment 6 *a*]). Later, when the bath temperature was raised to 31.2 C., a sensation of warmth was induced which after eleven minutes was only mild. The temperatures were then: 31.14 C., 30.20 C. and 28.49 C., i e., the gradients were reversed, and the temperature seemed neutral ten minutes later with temperatures of 30.84 C., 30.44 C. and 30.26 C.; the bath temperature had fallen slightly (from 0.1 to 0.2 C.) in the interval. Twenty minutes later, the sensation was on the "warm side of neutral" with temperatures of 30.92 C., 30.76 C. and 31.12 C.; at this time the temperature of the surface and at 0.8 mm. were no longer changing; that at 2.3 mm. was rising at 0.04 C. per minute. The gradient in the superficial layers was then reversed (0.20 C. per millimeter), and after a total of forty-five minutes, when the sensation had become again neutral, the temperatures were as recorded in the table (experiment 6 *c*) and the gradient was 0.025 C. per millimeter and again normal in direction. There appears to be greater correlation, in this and many such experiments, of the sensation with the spatial gradient than with the rate of temperature change in the dermis.

Since, however, changes in temperature of the bath altered gradients and these changes might be more significant during stasis, further experiments were conducted to determine the threshold change for sensation when the bath temperature was only altered slowly. Measurements of this type were carried out, for instance, in experiment 9. In

this experiment the initial temperatures in air were: surface of finger, 34.72 C., and 1.8 mm. deep to this, 35.11 C., i. e., a gradient of about 0.22 C. per millimeter. After fifteen minutes in a bath at 28.4 C., the temperatures were 29.18 C. and 30.66 C. (gradient 0.82 C. per millimeter), yet the sensation was neutral; after twenty-eight minutes, they were: bath, 28.45 C.; surface, 28.91 C.; at 1.8 mm., 29.64 C. (gradient 0.41 C. per millimeter), with the sensation still neutral. The bath temperature was then slowly raised (0.6 C at 0.26 C. per minute), with only fleeting sensations of slight warmth and coolness and without the subject being able to decide whether the bath had been warmed or cooled. A rise of 0.6 C. at 0.58 C. per minute gave a very definite sensation of warmth; the first detectable sensation occurred when the temperatures had been raised from: surface, 29.44 C.; at 1.8 mm., 30.38 C. (gradient 0.52 C. per millimeter) to: 29.51 C. and 30.38 C. (gradient 0.48 C. per millimeter) in 23 seconds; definite warmth when temperatures of 30.05 C. and 30.83 C. (gradient 0.43 C. per millimeter) had been reached in sixty-four seconds. When the gradients were estimated in this way from the changes at a depth of 1.8 mm., the sensations corresponded somewhat better with the rates of change on the surface than with the spatial gradients. In a series of such experiments with fingers only immersed, warmth was recognized when the rate of rise on the surface was from 0.1 to 0.3 C. per minute; coolness when the rate of fall was from 0.15 to 0.25 C. per minute. Experiments were conducted at temperature levels of 30 C. and 34 C.; the lower values for the thresholds for both cold and warmth were obtained at 34 C.

COMMENT

The experiments reported include thermocouple measurements of temperatures in the forearm, hand and finger. The conditions were somewhat different in the different localities. In the forearm and hand, the surface temperature was identical (within the accuracy of measurement) with that of the rapidly circulating water of the bath, except during the period of maximum reactive hyperemia, when the surface temperature might be raised or lowered a few hundredths of a degree. In the finger, however, the resting surface temperature might be definitely above that of the water (see table 3, experiment 8, where the experimental errors were probably minimal), and changes in surface temperature might be quite appreciable during reactive hyperemia. This difference may have resulted from the relatively high blood supply with direct arteriovenous channels of the finger,[8] though this area may also have been more protected from water currents. Fluctuations in surface

8. Krogh, August: Anatomy and Physiology of Capillaries, New Haven, Conn. Yale University Press, 1929, p. 101.

and deep temperatures in the fingers gave some evidence of the vasodilatation to cold described by Lewis.[9] On prolonged immersion in cold water, cool sensations often persisted longest on the lateral aspects of the roots of the fingers, suggesting that a higher level of blood flow modified sensation.

To induce sensation by varying the water temperature required much more rapid changes than occurred from incomplete control of the bath temperature; the threshold values for rate of change agreed fairly well with those reported by Gertz.[10] Sensations produced by altering the water temperature agreed somewhat better with spatial gradients than with rates of change of surface temperature, though no good agreement was found (or could be expected) with the average gradient for the dermal area; persistent sensations of warmth in baths appeared to be associated with reverse thermal gradients in the dermis, but sensations of very definite warmth might be caused by decreases in the normal gradients without reversal.

The immediate intensification of cold sensations produced in a cold bath by induction of stasis, however, must be associated with a decrease in tissue-blood vessel thermal gradients, and suggests that such a gradient is not the essential cause of sensation, and that Ebbecke's hypothesis cannot be accepted without modification.

There can be no doubt that the sensations produced during stasis and on release, when the arm is immersed in water at from 36.5 to 37.5 C., cannot be caused by changes in temperature, nor by differences in temperature between the tissues and inflowing blood, for the sensations may be intense when the temperature differences are minimal. One is forced to the conclusion that blood, which has been stagnant, has the capacity of inducing sensations of warmth when allowed to recirculate, and that the sensation produced is more intense (experiments 5 and 6) the longer the period of stasis, and the more asphyxial the blood. The sensations in the first of a series of periods of stasis tended to be somewhat more intense than in later ones, and this might mask this effect (experiments 5 and 7). If this is the case, stasis of the finger, which contains no striated muscle or other tissues with a high metabolism rate, should give less intense sensations of warmth than similar periods of stasis applied to the whole arm, for it must be remembered that in the latter case much of the blood must lie stagnant within vessels in muscular tissue, and that the blood supply to the skin is derived from vessels which leave the muscle and pierce the deep fascia. This difference in the intensity of warmth is clearly demonstrated in the data quoted in table 3. Though no data are given there

9. Lewis, T.: Heart **15**:177, 1930.

10. Gertz: Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **52**:1 and 105, 1921.

of experiments at rectal temperature levels, yet the same difference was detectable; thus, in one such experiment stasis of the finger alone for five minutes gave, on release, sensations "hot" and "warm," lasting in all for twenty-five seconds; stasis of the forearm for a similar period gave a "hot" sensation for thirty seconds and one of warmth lasting over two minutes. Sensations of warmth developing during stasis seem to have a similar origin, for they paralleled to some extent the development of cyanosis, and were not in evidence even after stasis for fifteen minutes at the lowest temperature levels, where metabolism was depressed. Yet with similar periods of stasis at higher temperature levels, the sensations began to fade toward the end of stasis, to be renewed rapidly after release, when the skin must be flooded with blood previously stagnant in muscle and likely to be even more asphyxial than that previously stagnant in the skin. Experimental evidence of a wave of increased acidity on release, though by no means conclusive, has already been advanced by us.[11] If, for simplicity of statement, an exaggerated acidity be supposed to be the metabolic product responsible for causing a sensation of warmth under these conditions, it would appear that it is a sudden change in acidity or a spatial gradient in acidity distribution that is involved, rather than the acidity level. If some such stimulus is concerned it must be capable of stimulating both warm and cold end-organs, for the sensations on release may be definitely "hot," even during stasis a "hot" element is possibly present, and the origin of "hot" sensations from simultaneous stimulation of warmth and cold seems now generally accepted (Burnett and Dallenbach,[12] Hahn [13]). Yet cold end-organs must be stimulated less readily, since definite cold or cool sensations have never been experienced during or after stasis either of the forearm or finger maintained at about 37 C. Warm sensations might, however, end abruptly and the subject might then indicate "? cool."

On the other hand, definite cold sensations have been experienced only when thermal gradients have been present. Release of warm blood into a cold finger or of relatively cool blood into a warm finger has given rise to such sensations, but when stasis has been applied to the whole arm they have been less evident in the former case, and absent or doubtful in the latter. Cooling blood at a constant carbon dioxide content shifts the p_H to the alkaline side and lowers carbon dioxide tension in vitro and in vivo (Austin and Cullen,[14] Bazett and Sribyatta [15]);

11. Bazett, H. C., and McGlone, B.: J. Physiol. **64**:393, 1928.
12. Burnett, N. C., and Dallenbach, K. M.: Am. J. Psychol. **38**:418, 1927.
13. Hahn, H.: Arch. f. d. ges Physiol. **217**:36, 1927.
14. Austin, J. H., and Cullen, G. E.: Hydrogen Ion Concentration in Health and Disease, Baltimore, Williams & Wilkins Company, 1926.
15. Bazett, H. C., and Sribyatta, L.: Am. J. Physiol. **85**:565, 1928.

if one supposes that the end-organs for cold may be stimulated by a wave of relatively alkaline blood, the results would be explicable and the diminution of sensation when the whole arm is involved in stasis might depend on the relative alkalinity of the blood being antagonized to a greater or less extent by acid metabolic products. The various combinations of cold, warm and hot sensations would then be explained if cold end-organs could be stimulated by a wave of either alkaline or acid blood, and warmth only by one of acid reaction but with a threshold below that for stimulation of cold end-organs. The fluctuating sensations often noted at temperatures of 30 C. might depend on an increased alkalinity of the blood by cooling, antagonized by acid metabolic products to varying degrees in the case of blood stagnant in the dermis (intermediate), fat (minimal) and muscle (maximal).

Similarly, modification of sensations by the duration of stasis would be readily explained. This phenomenon, which was first pointed out by Goldscheider and Hahn,[16] is well illustrated in experiment 6 *e* and *f*. One might suppose that with certain temperature-duration combinations the acidity and alkalinity changes might approximately balance, so preventing stimulation on release; on lengthening the duration of stasis the acidity should be increased, since the production of metabolic products has probably an approximately linear relationship to time, that of cooling an approximately exponential one. The facts that lengthening stasis did increase sensations of warmth in this case and that at a lower temperature level (experiment 4 *d*) stasis for fifteen minutes tended to give the same absence of sensations are in agreement with the hypothesis. It is perhaps noteworthy that the temperature at which the absence of sensation was most frequently noted was approximately that temperature at which Goldschmidt and Light[17] found minimal oxygen saturations of venous blood, which they interpreted as the result of considerable slowing of the circulation with only moderate depression of the metabolism. Recently, Achelis[18] described changes in chronaxia on release of stasis; he found a lengthening with stasis of long duration and sensations of heat on release; with stasis of short duration cold sensations were often experienced, and chronaxia values were shortened.

The sensations induced by stasis and its release have so far been discussed as though they were the result of sudden changes in either the acidity of end-organs, of blood in their neighborhood, or of spatial acid-alkali gradients. In favor of such a simple hypothesis are the sensations of warmth produced by contact with the skin of cool water

16. Goldscheider and Hahn (footnote 2, first reference).
17. Goldschmidt, S., and Light, A. B.: Am. J. Physiol. **73**:146, 1925.
18. Achelis, J. D.: Arch. f. d. ges. Physiol. **226**:212, 1930.

containing carbon dioxide (Liljestrand and Magnus [19]), or of carbon dioxide gas (Goldscheider and Ehrmann [20]), or produced by inhalation of gas rich in carbon dioxide (unpublished observations). Against such a hypothesis is the fact that end-organs for temperature appear sensitive to small changes in temperature, which would cause variations of only 0.1 mm. or less in the tension of carbon dioxide, yet gas mixtures containing high percentages of carbon dioxide (e. g., 10 per cent) are necessary to give definite sensations of warmth on inhalation. Even though abrupt changes in carbon dioxide tension induced in the lung must produce much more gradual changes peripherally, the discrepancy is considerable. Also other chemical substances may cause marked sensations of warmth; intravenous injection of small quantities of magnesium or calcium chloride produces this result (Keith [21]). Though, therefore, the general hypothesis of the stimulation of end-organs by metabolic products is not invalidated, and though an increase in hydrogen ion concentration may be the factor involved (e. g., as the result of injection of calcium chloride), yet variations in concentration of some other substance or ion may well be concerned, provided that its concentration is similarly affected by asphyxia and rise of temperature.

Sensations induced by variations in temperature appear to occur when the temperature levels within the dermis are stationary; they might depend either on stimulation of end-organs at some deeper level or on stimulation as the result of a thermal gradient between the tissues and blood. The former explanation seems to be excluded by the persistence indefinitely of warm sensations in a limb immersed in water at a temperature above that of the rectum (Gertz [10]), the latter would fail to explain the sudden increase in sensation of cold when stasis is induced in an arm immersed in cold water, since with the cessation of blood flow the tissue-blood gradient must be reduced. All sensations experienced could be explained as the result of cooling or warming of blood in the immediate neighborhood of the end-organs, whether this occurred as the result of a change in the temperature level of the whole area, of change in temperature of blood flowing across a thermal gradient, or of change in temperature of stagnant blood. Such changes in blood temperature might only act indirectly through the production of chemical changes or gradients. In this way differences in ionic potential across a membrane might be induced. Adaptation on prolonged exposure to a stimulus might in part depend on nervous processes, in part on local changes, for, when the temperature changes had extended sufficiently deeply to affect the temperature of the blood

19. Liljestrand, G., and Magnus, R.: Arch. f. d. ges. Physiol. **193**:527, 1922.
20. Goldscheider and Ehrmann: Arch. f. d. ges Physiol. **206**:303, 1924.
21. Keith, N.: Personal communication.

before it reached the neighborhood of the end-organ, i. e., when the normal thermal gradient had been reproduced, chemical adjustments between blood and tissue might occur before the blood reached the end-organ, so that the blood no longer acted as a stimulus. More precise statement, even as a tentative hypothesis, seems at present unwarranted. The most plausible explanation appears therefore to be some modification of Ebbecke's hypothesis, and the importance of the circulating blood and of thermal gradients is substantiated, even though his hypothesis needs to be considerably modified. The results obtained are also in fair agreement with those we have obtained on stimulation of cold and warm spots in the prepuce, data now in course of publication in detail.

CONCLUSIONS

1. Measurements have been made of the temperature changes in the forearm, hand or fingers on release of stasis, both with the limb in air and immersed in water at varying temperature, and the values obtained have been compared with the sensations induced so as to test Ebbecke's hypothesis of the origin of temperature sensations. Comparison has also been made of the effects of stasis of one finger only and of that of the whole forearm.

2. Sensations of intense warmth or heat were found to be induced on release of stasis in a limb maintained before, during and after stasis in a bath at the blood temperature level, so that on release no changes in temperature occurred, and no thermal gradient was established. These sensations were found to vary roughly according to the probable acidity of blood flowing into the dermal area, to be more intense with long periods of stasis than with shorter, and greater with stasis of the whole forearm than with that of a finger only. They are considered to be elicited by a chemical stimulus (acting probably through the establishment of a chemical gradient), derived from metabolic processes, particularly in muscle tissue, and by a substance that varies in concentration both during asphyxia and as the result of temperature changes in a manner similar to that of acid. The possibility of acid itself being the stimulus is discussed but is considered somewhat improbable.

3. The sensations at other temperatures are considered as caused by chemical changes of the same type antagonized to a greater or less extent by opposite chemical changes induced by thermal differences.

4. Some data are presented of the thermal changes and gradients in the dermis associated with temperature sensations induced by varying the bath temperature. They are considered to be most readily explained by some modification of Ebbecke's hypothesis.

5. Tingling sensations occurring during stasis and on its release are shown to originate peripherally in the area exposed to stasis and to be

modified by the temperature of this part; they are not dependent on pressure on the nerve trunks.

APPENDIX

Tingling was noted during stasis and following its release in these experiments, and a brief report has appeared (Bazett and McGlone [22]). The conclusion was reached that these sensations were of chemical origin and peripherally induced. The latter conclusion was drawn because of the marked effect of peripheral temperature changes. The recent work of Lewis, Pickering and Rothschild [23] indicates that tingling sensations following release are induced in the nerve trunks within the area of compression; their work may appear to throw doubt on the conclusions drawn by us in regard to the causation of both tingling and temperature sensations.

We assumed (1931) that the temperature of the nerve trunks above the water level would not show any considerable change. Direct measurement indicates that the changes are much greater than we anticipated. The temperature beneath the fascia 12 mm. from the surface on the inner aspect of the biceps muscle, close to the main vascular and nerve trunks of the upper part of the arm (junction of middle and lower thirds), was 36 C. with the arm in air. In another experiment, after the forearm had been in a bath at 34.9 C. for fifty minutes, a temperature of 35.8 C. was observed. On the first occasion a fall of 2.4 C. resulted at this depth from immersion of the forearm only in water at 15 C. for fifteen minutes; on the second occasion a fall of 4.5 C. resulted from a similar immersion for fifty-two minutes. Therefore, temperature changes in the nerve trunks are not negligible; some evidence was obtained that if the upper part of the arm became wet (as with a wet armlet), the temperature change was somewhat greater.

Experiments of the type employed by Lewis and his associates were conducted; two armlets were employed, one on the upper part of the arm and one on the forearm; the lower was inflated before the release of the upper. Pressures of from 150 to 180 mm. were employed for periods of fifteen minutes for the upper armlet and from five to six for the lower. Lewis' observations as to the distribution of tingling were fully confirmed, but the temperature sensations had quite a different distribution. Definite sensations of temperature were limited to the areas involved in reactive hyperemia, and consequently were not referred to the area below the lower armlet on release of the upper. However, in some subjects, slight sensations of warmth were experienced in the hand and fingers even on release of the upper armlet, while

22. Bazett, H. C., and McGlone, B.: Proc. Soc. Exper. Biol. & Med. **29**:87, 1931.

23. Lewis, Thomas; Pickering, G. W., and Rothschild, Paul: Heart **16**:1, 1931.

the lower armlet was still compressed, but in at least one of these subjects definite changes in color could be detected in the fingers on release of the upper armlet, and collateral circulation through bones cannot have been negligible. In no case were the sensations of temperature in the fingers and hand on release of the upper armlet in any way comparable in degree with the much more intense sensations experienced on the release of the lower armlet.

Though the sensations of tingling on release appear to be generated in the nerve fibers under and distal to the compressing armlet, this does not appear to be the case with temperature, and the peripheral origin of such sensations assumed in this paper is justifiable. The similar origin of sensations of tingling assumed in our previous preliminary report is not substantiated as far as the phenomena of release are concerned; the situation in relation to tingling during stasis is much less clear.

NEUROLOGIC ASPECTS OF PRIMARY ANEMIA

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MINNEAPOLIS

This paper has been undertaken with the hope of adding further facts to an already well established knowledge of the neurologic involvement in primary anemia. Considerable material has been accumulated since 1886, when Lietheim [1] first described what are known today to be symptoms of subacute combined degeneration of the spinal cord. In spite of this accumulated material, many conflicting opinions exist about points that probably can be established only when larger groups of cases are studied.

This study is based on the routine neurologic examination in 117 cases of proved primary anemia at the Nicollet Clinic of Minneapolis and in seventy-two similar cases at the Minneapolis General Hospital. The examinations have been made during a period of seven years. The patients were not referred for examination because of marked neurologic signs or symptoms, but as a matter of routine hospital and clinic practice. Naturally, many patients were not examined by the neurologist because they were moribund, because of lack of cooperation on the part of the patient or for other reasons, but no effort to select the cases was made. The study can, therefore, be regarded as representative of an average group of such cases in this locality.

A series of tabulations can best present the observations in the total series. No striking differences existed between the clinic cases and the hospital cases. The two series will, therefore, be presented together. The tabulations cover the points of the examination and are followed by a discussion:

From the Nicollet Clinic, Department of Neurology.

Read before the Section on Nervous and Mental Diseases at the Eighty-Second Annual Session of the American Medical Association, Philadelphia, June 11, 1931.

1. Lietheim: Zur Kenntnis der perniciösen Anämie, Verhandl. d. Cong. f. inn Med. **11**:84, 1887.

Involvement of the Cranial Nerves.—Involvement of the cranial nerves was noted as follows:

Central area of impaired vision with peripheral vision good	1
Fresh hemorrhage in fundus	3
Vision impaired, unilateral	1
Pupillary irregularity	12
Ptosis, bilateral and unequal	1
Impaired convergence	1
Nystagmus	1
Hearing lost: bilateral	2
unilateral	1
impaired	3
Smell impaired	3
Tremor of the tongue	1
Slurring speech	1

Much of the involvement of the cranial nerves might be regarded as accidental, and in most instances it bears no relation to the anemia. In the case of loss or disturbances of hearing and smell, however, it would seem that there was some basis for regarding them as part of the neurologic picture caused by involvement of the central nervous system, since the loss was associated by history with the development of other symptoms. It is my opinion that cranial nerve changes, when present, are of little or no early diagnostic significance. Certainly there is no predominantly characteristic involvement demonstrated by this or by other series of cases that have been reported.

Coordination.—Impairment of coordinate movements was noted as follows:

	Impaired
Arms	20
Legs	76
Romberg sign	79

Often in interpreting the degree or presence of impaired coordinate movements one is apt to misinterpret muscular weakness for central involvement. This must be kept in mind and allowance made for it. It is not unusual to see a marked improvement in this field under the influence of improved nutritional tone, this occurring even before the advent of liver therapy. It is to be noted that the lower extremities are more often and more profoundly involved than the upper, which is in keeping with the reflex and sensory changes.

Gait.—Various disturbances in gait were present, as follows:

Paralyzed	5
Learned to walk	2
No improvement	3
Ataxic	29
Spastic	13
Spastic-ataxic	5

Disturbances of gait are relatively common and must also be regarded as possible manifestations of weakness, as well as of spasticity and incoordination and loss of deep sensibility. Certainly, disturbances of gait are exaggerated by such weakness and tend to improve with general improvement.

Involvement of the Sphincters.—Involvement of the sphincters was noted as follows:

	Rectal	Vesical
Retention		3
Incontinence	2	8

Involvement of the sphincters occurs as part of the picture in extreme involvement of the cord. One might expect it to occur more often. It varies with the general condition of the patient.

Deep Reflexes.—The deep reflexes were graded as follows:

	Plus 2	Plus 3	Plus 4	Minus 2	Minus 3	Minus 4
Biceps	30	6			2	2
Patellar	30	32	2	19	12	32
Achilles	31	16	1	8	12	55

All deep reflexes normal.....18

Plantar: positive, 28

Abdominal: Increased, 0; diminished, 18; absent, 26; unequal, 2.

In grading reflex changes the scale of 4 was used, in which zero represents normal, +4, an increase to the point of well sustained clonus and —4, a total absence. Normal and —1 changes were not recorded.

Few cases have shown constantly normal reflexes. Except for a moderate increase, the upper extremities were not affected, although in cases in which peripheral neuritis was present decrease or loss was encountered. At least, peripheral neuritis was present in the four cases of this series. In the lower extremities all degrees of change and variation may be found, but here, as in other observations, the distal portion of the lower extremities was more constantly and severely involved. As suggested by these figures and by clinical observation, the tendency is toward a loss of the achilles reflex, although marked spasticity may have been present at some time, or persistent over a period of time. Later discussion will bring out the variability of these findings.

Changes in the plantar reflexes form a part of the picture of lateral cord involvement. They may be positive when the deep reflexes are absent.

The abdominal reflexes are frequently absent or diminished, and have come to be looked for as a part of the typical picture.

Paresthesia.—A history of paresthesias was noted in 133 patients, but data were lacking in many instances. All sorts of descriptions of these subjective sensory disturbances were offered, but the most common was

that of "pins and needles." The usual distribution involved the fingers and toes, but in several instances the entire body was involved. In several the hips and buttocks alone were affected. A unilateral distribution was not unusual. The condition varied from a mild disturbance to that of actual pain, and occasionally the fingers became so sensitive that touching objects was unbearable. Deep pain (not cramp) was also frequently described.

I am of the opinion that these paresthesias bear no relation to the cord changes. They may occur before cord changes can be demonstrated, and may be totally absent in cases showing profound posterolateral sclerosis. They tend to disappear and reappear without proportional changes occurring in the central nervous system. They often appear early in the fingers, and may persist there after they have disappeared in the toes, but seldom does one find reflex or deep sensory changes in the arms.

Sensory Disturbances.—Various sensory disturbances were noted, as follows:

	Decreased	Absent	Increased
Temperature	51	2	3
Pain		(Segmental	2
Touch		level)	
Calf	6	2	12
Joint	15	20	

Occasional impairment of superficial sensation over the back and thorax only was noted, but usually the impairment was present in the extremities and varied considerably in degree. The distal parts of the extremities were most affected, the impairment fading into normal as the upper portions were approached. In two cases of this series there was a distinct segmental level to pain, touch and temperature. Dissociation was not unusual, touch being perceived and pain being disturbed. The reverse was sometimes true.

In certain cases it has seemed that the impairment to light touch and pain was proportional to the degree of paresthesia present, improving as the tingling sensation disappeared. In other cases there was no apparent relationship.

A differential diagnosis between cord tumor and combined sclerosis should not be difficult, but when segmental levels such as were noted here occur, a question may arise. Figure 1 shows the sensory loss in such a case.

It is noteworthy that deep muscle sense is not frequently disturbed. In fact, it is often acute. On the other hand, joint sense is often impaired or absent. Owing to the many factors that influence this test, such as unequal pressure of the examiner's fingers, friction of the fingers

on the skin, stretching of the skin about the patient's toe, on the dorsum or sole of the patient's foot, or friction of toe against toe, I have not regarded this test as very reliable where superficial sensation and muscle sense were intact.

Of all the tests that aid in the diagnosis of posterolateral sclerosis, the quantitative examination of the vibratory sensation is, I believe, the most reliable. Many observers [2] have noted the early disturbance of deep sensibility.

In 1925, I published [3] a detailed study of the quantitative vibratory sensation as made with Symns' quantitative fork in normal and pathologic

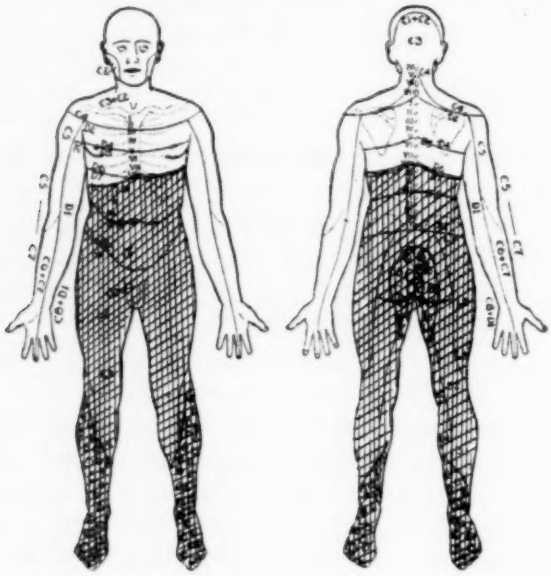

Fig. 1.—Tactile sensation and pain sensation were graded —4, as diagramed. Vibratory sensation as determined with a fork was absent in the lower extremities.

cases. This fork is made by Downs Brothers, of London, and is standardized against the fork used by Symns.[4] It is of from 108 to 76

2. Dejerine, A., and Mouzon, J.: Rev. neurol. **22**:382 and 388, 1914. Dejerine, J.: Compt. rend. Soc. de biol. **75**:554, 1913. Dejerine, J., and Jumentié, J.: Rev. neurol. **22**:273, 1914. Cadwalader, W. B.: Diagnosis of Subacute Combined Sclerosis of the Spinal Cord Associated with Severe Anemia, J. A. M. A. **66**:2035 (June 17) 1916. Woltmann (footnote 5). Hamilton, A. S., and Nixon, C. E.: Sensory Changes in Subacute Combined Degeneration of Pernicious Anemia, Arch. Neurol. & Psychiat. **6**:1 (July) 1921.

3. Ahrens, R. S.: Vibratory Sensation, Arch. Neurol. & Psychiat. **14**:793 (Dec.) 1925.

4. Symns, J. L. M.: Brit. M. J. **1**:539, 1912; Guy's Hosp. Rep. **66**:120, 1912; Quart. J. Med. **11**:33, 1917.

double vibrations. The ends of the prongs are fitted with metal lugs that overlap, one of which is notched so that on holding it against the light when it is vibrating one is able to determine when a fixed amplitude of vibration has been reached by watching the notches disappear. Starting always at this fixed amplitude, one can determine with a stop watch and the cooperation of the patient how long the vibration is felt. In 100 normal cases, the shortest time the vibration was felt over the toe and internal malleoli was nine seconds; the longest time was twenty-nine seconds, and in the majority it was between twelve and twenty-one seconds.

In that first report I reviewed seventeen cases of primary anemia in which not one presented a normal quantitative vibratory sensation. No case showed an absence or impairment in the upper extremities alone No case showed a total loss in both hands and feet.

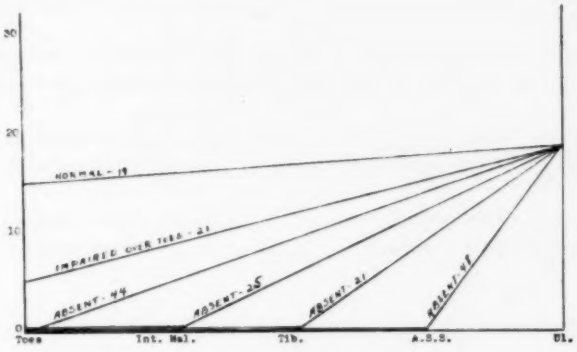

Fig. 2.—Curve of quantitative vibratory sensation, with the number of cases.

In accordance with other observers, I pointed out that if a curve of the quantitative vibratory sensation studies in pernicious anemia was plotted, using the time in seconds on the vertical line and the point examined as the base, a rising curve would be obtained with the greatest impairment in the distal parts of the lower extremities and an approach to normal as the upper portions of the body were reached.

In the present report the same fact maintains. Such a curve is presented in figure 2.

It is to be noted that: 1. Of 178 patients examined, 159, or 89.3 per cent, showed some degree of impairment. This is in keeping with Woltmann's [5] figures, which were 82.4 per cent diminished and 33.6 per cent absent in 150 cases (using a C fork of 256 vibrations), and those of

5. Woltmann, H. W.: The Nervous Symptoms in Pernicious Anemia: An Analysis of 150 Cases, Am. J. M. Sc. **157**:400, 1919.

Cases in Which Treatment Was Given *

Case	Date	Deep Reflexes: Biceps	Deep Reflexes: Knee	Deep Reflexes: Achilles	Superficial Reflexes: Plantar	Superficial Reflexes: Abdominal	Vibratory Sense, Number of Seconds: Toe	Vibratory Sense, Number of Seconds: Inner Malleolus	Vibratory Sense, Number of Seconds: Tibia	Vibratory Sense, Number of Seconds: Anterior Superior Spine	Vibratory Sense, Number of Seconds: Ulna	Coordination	Gait	Hemoglobin	Red Blood Cells
1	3/31/26	0 0	−3 −3	−4 −4	0	0	0 0	0 0	0 0	3 3	16 18	0 0	...	76	4,210,000
	6/22/26	0 0	−2 −2	−3 −3	0	0	0 0	0 0	0 0	0 0	17 18	−4 −4	...	80	4,820,000
2	6/15/26	0 0	+1 +1	0 0	0	0	0 0	0 0	0 0	0 0	C.F.	0 0	...	76	3,000,000
	1/10/27	0 0	0 0	−2 −2	0	0	0 0	0 0	0 0	0 0	17 16	FN −1 −1 HK −2 −2	...	76	3,990,000
3	10/ 5/26	+2 +2	−2 −2	−4 −4	0	0	0 0	0 0	0 0	0 0	20 22	FN −1 −1 HK −3 −3	At +3	68	2,520,000
	11/19/26	0 0	−3 −3	−4 −4	0	0	0 0	0 0	0 0	0 0	18 20	HK −2 −2	...	77	2,830,000
	4/ 2/28	−1 −1	−2 −2	−4 −4	0	0	0 0	0 0	0 0	0 0	20 20	HK −2 −2	...	76	4,280,000
	11/14/29	.....	−1 −1	−4 −4	0	0	0 0	0 0	0 0	0 0	19 18	HK −1 −1	Normal	76	4,310,000
4	10/26/28	−1 −1	−4 −4	−4 −4	0	0	0 0	0 0	0 0	0 0	16 15	HK −3 −3	At +3	77	3,790,000
	12/28/29	.....	−2 −2	−4 −4	0	0	0 0	0 0	0 0	0 0	18 17	HK −3 −3	At +3	89	5,140,000
5	7/18/27	0 0	−2 −2	−4 −2	0	0	0 0	0 0	5 8	10 9	16 18	0	0	60	2,320,000
	5/11/28	0 0	−2 −3	−4 −4	0	0	0 0	0 0	4 7	7 7	20 20	0	0	79	4,210,000
6	6/28/29	+1 +1	+2 +2	+1 +1	0	0	0 0	6 0	10 5	10 12	19 22	0	0	46	2,200,000
	7/ 9/30	0 0	+3 +2	+2 +2	0	0	0 0	12 6	20 16	12 13	20 20	0	0	80	3,900,000
7	4/ 4/28	0 0	+2 +2	+2 +2	0	0			Normal			0	0	70	3,660,000
	2/27/29	0 0	+1 +2	+2 +2	0	0			Normal			0	0	85	4,360,000
	3/17/31	0 0	+1 +1	+1 +2	0	0	15 15	14 16	17 18	12 13	20 22	0	0	90	4,530,000
8	4/20/30	0 0	+1 +1	+2 +2	0	0	0 0	10 12	15 14	16 14	20 21	0	0	51	3,300,000
	3/17/31	0 0	0 0	0 0	0	0	0 0	10 0	14 14	14 15	24 23	0	0	46	3,000,000
9	8/27/30	0 2	−4 −4	−4 −4	0 +	0			Not tested			0	At +2	111	4,560,000
	1/21/31	+2 −2	−4 −2	−4 −4	0 0	0	10 12	12 15	15 15	12 13	20 20	HK −2 −2	0	90	4,300,000
	3/24/31	+1 −3	−3 −3	−4 −4	0 0	0	9 12	12 12	15 16	10 10	19 21	0	0	98	4,360,000
10	11/11/29	0 0	0 0	0 0	? 0	0	12 14	16 14	15 15	15 15	20 21	0	0	40	2,800,000
	3/17/31	0 0	+2 +3	+2 +2	....	...	14 14	16 16	18 16	14 13	22 20	0	0	98	4,250,000
11	1925	0 0	+2 +2	+2 +2	0	0	10 11	11 11	13 13	16 14	21 19	0	0	45	3,340,000
	1928	0 0	+1 −0	0 0	0	0	0 0	5 7	10 10	10 9	18 20	0	0	70	4,205,000
	1930	−2 −1	−4 −4	−4 −4	0	0	0 0	0 0	0 0	5 7	17 18	HK −3	At −3	85	4,700,000
	1931	−1 −1	−4 −4	−4 −4	0	0	0 0	0 0	0 0	0 0	18 18	HK −3	At −3	80	4,530,000
12	1/ 5/26	+1 +1	+1 −1	0 −2	0	0	15 0	17 13	20 18	12 15	22 20	HK −1 −2	0	79	3,760,000
	1/ 7/27	+1 +1	−2 −2	−1 −1	0	0	0 0	0 0	4 3	9 7	18 20	HK −2 −3	At +1	79	3,320,000
	1/ 4/28	0 0	−4 −4	−4 −4	0	0	0 0	0 0	0 0	0 0	20 21	HK −4 −4	At +2	74	4,170,000
13	4/22/26	+2 +2	+3 +3	+3 +3	0	0	10 5	12 14	16 18	15 17	23 20	HK −1 −2	0	40	3,280,000
	10/12/26	0 0	+2 +2	+1 −1	0	0	8 0	10 10	10 10	18 16	20 18	HK −2 −2	0	53	3,660,000
	1/31/28	+1 +1	+2 +2	0 0	0	0	0 0	12 10	10 12	15 14	19 21	HK −3 −3	0	41	3,070,000
14	3/ 9/27	+1 +1	+2 +2	+1 +1	0	0	9 4	12 6	14 7	15 14	18 20	0	0	63	2,800,000
	3/14/29	+2 +2	+3 +3	+3 +3	+ +	0	0 0	0 0	0 0	0 0	20 22	HK −4 −4	At	76	2,990,000
15	3/16/28	+1 +1	0 0	−1 −1	0	−4	0 0	0 0	7 6	12 15	18 20	0	..	33	1,530,000
	5/ 2/28	+1 +1	−2 −2	−2 −2	0	...	0 0	0 0	6 6	7 9	20 22	HK −2	..	74	3,070,000
	3/23/31	0 0	+3 +3	0 0	0	−4	0 0	0 0	0 0	0 0	18 17	FN −2 HK −4	At + 3	84	4,540,000
16	6/18/28	+1 +1	+2 +2	0 0	0 +	...	0 0	0 0	0 0	0 0	23 25	HK −2	0	80	3,360,000
	4/10/31	0 0	+2 +2	−3 −3	0 +	...	0 0	0 0	0 0	0 0	20 19	0	0	85	3,980,000

17	9/20/27	+2 +2	+4 +4	0 0	0	0	0 0	0 0	0 0	0 0	16	18	0	Sp +3 At	66	3,320,000
	3/14/28	0 0	+3 +3	+2 +2	+ +	0	0 0	0 0	0 0	0 0	20	18	HK —4	Sp At +3	63	3,610,000
18	3/ 1/28	0 0	+1 +1	+1 +1	0 0	0	8 8	10 10	12 10	19 9	23	20	0	0	47	2,270,000
	3/26/31	0 0	+3 +4	+4 +4	+ +	0	0 0	0 0	0 0	0 0	18	20	HK —4	Wh. Ch.	100	5,300,000
19	3/18/30	0 0	+10	—40	0	0	0 0	6 5	8 9	10 10	24	22	0	0	30	1,450,000
	7/ 1/30	0 0	0 0	0 0	0	0	0 0	0 0	0 5	9 8	20	21	0	0	50	3,000,000
20	6/25/28	0 0	+3 +3	+3 +3	+ +	—4	0 0	0 0	0 0	0 0	20	18	HK —3 —3	..	45	2,360,000
	4/ 7/31	+2 +2	+4 +4	+4 +4	+ +	—4	0 0	0 0	0 0	0 0	20	20	HK —4 —4	Sp At	80	4,304,000
21	11/27/28	+1 +1	0 0	—4 —4	0	0	0 0	0 0	0 0	0 0	24	23	HK —3	..	34	1,400,000
	9/16/29	0 0	—2 —2	—4 —4	0	0	0 0	0 0	0 0	0 0	19	20	HK —3	..	92	4,720,000
	3/17/31	0 0	—3 —3	—4 —4	0	0	0 0	0 0	0 0	0 0	23	22	HK —3	At +3	100	4,930,000
22	4/19/29	0 0	—4 —4	—4 —4	0	0	0 0	8 6	10 11	12 10	18	18	HK —2 —2	At +3	31	1,630,000
	10/ 2/29	0 0	—4 —4	—4 —4	0	0	0 0	7 0	12 5	12 14	21	21	HK —2 —2	0	92	4,300,000
	3/24/31	+2 +2	+3 +3	+3 +3	+ +	0	0 0	5 0	10 0	9 5	20	18	HK —2 —2	Sp +3	115	4,500,000
23	3/25/29	+2 +2	—2 —2	—4 —4	0 0	0	0 0	0 0	4 6	10 8	20	22	HK —3	At +2	60	3,330,000
	9/10/30	0 0	+1 +1	+4 +4	+ +	0	0 0	0 0	0 0	0 0	18	19	HK —4	Sp At	84	3,850,000
	3/ 3/31	0 0	—4 —4	—4 —4	0 0	0	0 0	0 0	0 0	0 0	20	22	HK —3	At	90	4,600,000
24	2/10/28	+1 +1	+1 +1	0 0	0	—4	0 0	6 8	8 7	10 11	23	20	HK —3	0	36	183,000
	3/24/31	0 0	+2 +2	—3 —3	0	0	0 0	4 0	8 5	10 12	20	20	HK —2	0	80	4,660,000
25	6/25/28	0 0	+3 +3	+2 +2	+ +	0	0 0	0 0	0 0	0 0	20	18	HK —4 —4	Par	36	1,690,000
	6/ 8/29	0 0	+3 +3	+3 +3	+ +	0	0 0	0 0	0 0	0 0	18	20	HK —4	Sp At	85	4,900,000
	10/12/30	0 0	+3 +3	+4 +4	+ +	0	0 0	0 0	0 0	0 0	20	22	HK —4	Sp At	90	5,000,000
	5/ 7/31	0 0	+3 +3	+4 +4	+ +	0	0 0	0 0	0 0	0 0	22	22	HK —4	Sp At	93	5,300,000
26	9/21/26	Neurologically normal, mentally disturbed; considered to be a case of psychoneurosis, but blood suggestive of pernicious anemia; no achlorhydria													73	3,120,000
	3/24/27	Sore tongue and spleen palpable; mental condition same—several fights—not following diet													78	3,740,000
	10/24/24	0 0	+2 +2	+2 +2	0 0	0	10 11	12 12	12 12	8 8	13	14	0	0	74	3,680,000
	12/30/30	0 0	+2 +2	0 0	0 0	0	0 0	8 7	10 12	9 8	18	17	0	0	70	3,960,000
	5/ 1/31	0 0	0 0	—3 —3	0 0	0	0 0	0 0	0 0	5 7	16	14	—2	0	80	4,860,000
27	11/15/26	0 0	+2 +2	+2 +2	0 0	0	14 15	16 15	17 17	14 10	20	18	0	0	40	1,860,000
	11/ 8/28	0 0	0 0	—2 —2	0 0	0	12 14	13 13	13 14	13 11	19	18	0	0	86	5,060,000
	5/ 3/31	0 0	0 0	0 0	0 0	0	10 9	11 12	12 12	14 12	21	19	0	0	83	4,540,000
28	3/24/31	0 0	—4 —2	—4 —4	0 0	0	0 0	3 0	4 0	6 0	18	20	0	0	66	3,690,000
	4/18/31	0 0	—4 —2	—4 —4	0 0	0	0 0	3 0	3 0	0 0	20	19	0	0	77	4,230,000
29	4/ 6/30	0 0	—4 —4	—4 —4	+ +	0	0 0	0 0	0 0	0 0	18	20	—4 HK	Sp At	60	2,900,000
	3/ 5/31	0 0	—4 —4	—4 —4	+ +	0	0 0	0 0	0 0	0 0	20	20	—4 HK	Sp At	80	4,200,000
30	12/27/27	0 0	+2 +2	+2 +2	0 0	0	0 0	5 4	5 5	10 12	20	21	HK —2 —2	0	47	2,020,000
	1/ 7/28	.....	.....	.....	....	...	.....	.....	.....	.....	.....	.....	.....	..	72	2,530,000
	4/ 8/31	0 0	+2 +2	+3 +3	0 0	0	0 0	0 0	0 0	0 0	20	19	HK —3 —3	0	..	.....
31	6/11/28	0 0	+2 +1	0 0	0 0	0	0 0	12 14	14 15	12 12	25	20	HK —1 —2	0	88	3,300,000
	5/ 9/29	0 0	0 0	0 0	0 0	0	0 0	0 0	8 7	9 11	18	19	HK 0 0	0	85	4,300,000
32	6/11/24	0 0	0 0	0 0	0 0	0	0 0	0 0	9 11	9 9	18	20	HK —2 —2	0	70	2,930,000
	3/12/28	0 0	—2 —2	—4 —4	+ +	0	0 0	0 0	0 0	5 0	20	20	HK —4 —4	0	63	2,930,000
	4/ 8/31	0 0	—4 —4	—4 —4	0 0	0	0 0	0 0	0 0	0 0	16	19	HK —4	At	85	3,800,000
33	7/ 5/27	0 0	—4 —4	—4 —4	+ +	0	0 0	0 0	0 0	0 0	16	16	HK —4 —4	Sp At	40	2,100,000
	5/10/31	0 0	—4 —4	—4 —4	+ +	0	0 0	0 0	0 0	0 0	17	18	HK —4 —4	..	72	3,650,000
34	4/17/30	+1 +1	—4 —4	—4 —4	+ 0	0	0 0	0 0	0 0	0 0	18	19	HK —4 —4	At	40	1,970,000
	5/12/31	+1 +1	—4 —4	—4 —4	+ 0	0	0 0	0 0	0 0	0 0	20	20	HK —4	Imp	90	4,610,000

* In the table, HK indicates the heel-to-knee test; FN, the finger-to-nose test; At, ataxic; Nor, normal; Sp, spastic; Imp, improved; C.F., tuning fork; Wh. Ch., wheel chair, and Par, paralyzed lower extremities.

Hamilton and Nixon,[6] who reported impairment or absence in 87.5 per cent of forty cases.

2. Impairment in the upper extremity is relatively rare and is of no diagnostic importance, since it occurs only after profound changes have developed in the lower extremities. Patients who have shown an absence of vibratory sensation in the upper extremities have also exhibited a marked peripheral neuritis and a fulminating neurologic involvement.

3. Frequently the only positive or significant discovery in the examination is a quantitative impairment or loss of vibratory sensation over the first toes. This occurred in sixty-one patients. When it is noted, I expect to find a rising curve approaching normal as I ascend the lower extremities. In six of the cases attention was directed to the correct diagnosis because of this loss. Even a quantitative reduction in vibratory sensation over the toes with a rising curve should lead to a consideration of early cord changes and careful ruling out of primary anemia as the cause.

Mental Changes.—The following mental changes were noted:

Attacks of profound stupor	5
Slow cerebration and impaired memory	25
Anxiety	1
Depression, fearful of insanity	2
Marked nameless fear—quit a good position	1
Delusions:	
Grandiose	1
Body odor	1
Euphoric	1
Strings around legs	1
Persecution	4
Change of personality—irritable	2
Confusion—disoriented	5

Mental changes are common and may vary from mild personality changes to a definite, well organized, delusional psychosis. The most common disturbance is one of slowed cerebration. Many patients complain of loss of memory (impaired attention) and irritability. Depressed states are noted, and anxiety of varying degree may develop. Delusional trends may be fixed and well organized. One patient became difficult to handle because of her belief that relatives were placing needles in her bed. Another patient insisted that someone threw strings around his legs, causing him to fall. A third patient, divorced by his wife, caused considerable trouble by accusing the judge in the case of being friendly with his wife and scheming with her to obtain his property. Investigation revealed abnormal mental reactions, directed first toward his wife and leading to the divorce, and later directed toward the judge. Euphoria

6. Hamilton and Nixon (footnote 2).

is not unusual, as in the case of one patient who wasted almost all of his estate on grandiose schemes. Another patient, a banker, had been sentenced to prison for the misappropriation of bank funds before his true mental condition was recognized. When admitted to the hospital, he wanted to present everyone with their choice of an automobile.

From a medicolegal standpoint such psychotic phases should be regarded as important. A man, brought to the General Hospital from the workhouse, presented a well developed anemia and early cord changes. In addition, he showed marked impairment of judgment and lack of insight concerning his physical condition and an indifference to his criminal record. His past life had been well ordered and industrious, and as his mental condition improved under liver treatment it became evident that his forgeries were the product of a disturbed brain.

I will make no attempt to discuss this phase of the neurologic picture. Woltmann [7] presented a most complete review of the literature and the results of histologic study in seven cases in which demonstrable pathology comparable with that occurring in the cord was found in both the gray and the white substance. Whether these pathologic changes are responsible for the mental changes is still doubtful. Further study should be directed toward this phase of the picture.

COMMENT

Of a total series of 189 cases, 185, or 97.77 per cent, showed evidence of cord involvement. In arriving at this figure, only deep reflex change, of a plus or minus 2 degrees or greater, and quantitative loss of vibratory sensation in the lower extremities were considered. Paresthesias and other subjective disturbances have not been used in determining the percentage given.

The quantitative absence of vibratory sensation in the toes has been regarded as a positive sign. Justification for this lies in the fact that such cases were otherwise proved to be pernicious anemia, and at a reexamination almost always showed further evidence of cord involvement.

Two cases in this series showed a quantitative diminution in the vibratory sensation over the toes and internal malleoli, the response approaching normal as the upper parts were reached. In both cases deep reflex changes were also present, but if the reflexes had been normal, I would have considered such a reduction in vibratory sensation as positive evidence of early posterior column involvement.

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7. Woltmann, H. W.: Brain Changes Associated with Pernicious Anemia, Arch. Int. Med. **21**:791 (June) 1918.

A résumé of the preceding tabulations gives the following percentages:

Coordination disturbed:	
Arms	10.6
Legs	40.2
Romberg positive	41.7
Gait disturbance	27.9
Deep reflexes increased or diminished	90.4
Vibratory sensation reduced	89.3
Superficial sensation disturbed	26.6

The percentage would have been lower if cord involvement had not developed while under observation (i. e., if original negative findings only were considered). They would also have been lower if an ordinary tuning fork had been used instead of a quantitative determination of vibratory sensation being made.

These figures compare favorably with those of Woltmann,[5] who reported 80.6 per cent of 150 cases as showing cord involvement; of Hamilton and Nixon,[6] who reported from 75 to 80 per cent, and of Minnich,[8] who reported pathologic findings in 77 per cent of cords from patients with primary anemia. Very low percentages have been reported, but it would seem that with a wide knowledge of the subject and keener interest, together with more refined means of examination estimates should more closely approach each other. However, in so recent a book as that of Davidson and Guilland,[9] in which the subject is most adequately discussed in some respects, one finds the emphatic statements that only 5 per cent of all cases show cord symptoms; that the high figures of some authors were obtained by the inclusion of paresthesias, and that to the 5 per cent cited must be added "The cases which go to a neurologist and which may never be recognized as cases of pernicious anemia."

An improved neurologic technic would, I am sure, change the figures, and a more careful survey of the literature would convince these authors that paresthesias are not used in arriving at the percentage of cord involvement; as for the latter statement, no neurologist worthy of the title is missing the diagnosis today.

Given the opportunity to reexamine these patients at stated intervals, I believe that all cases of pernicous anemia would show evidence of subacute combined degeneration of the spinal cord at some time during the progress of the disease.

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8. Minnich, W.: Zur Kenntnis der in Verlauf der perniciösen Anämie beobachteten Spinalerkrankungen, Ztschr. f. klin. Med. **21**:25 and 264, 1893; **22**:60, 1893.

9. Davidson, L. S. P., and Guilland, G. L.: Pernicious Anemia, St. Louis, C. V. Mosby Company, 1930.

Of my series, thirty-four have been treated with liver or liver extract and have been under observation for from several months to three years. I have personally examined and reexamined these patients, many of them several times at variable intervals. An analysis of the tabulated data presented in detail at the end of this article develops some interesting points. Of the thirty-four cases, two (cases 29 and 34) remained stationary, each over a period of eleven months. Both showed an absence of deep reflexes in the lower extremities and a quantitative loss of vibratory sensation up to and including the anterior sacral spines. The hemoglobin and red cell count responded well to liver therapy, but the cord symptoms have shown no change. Of the remaining thirty-two cases, all have shown changes of one form or another.

Deep Reflexes	Knee Jerk	Achilles Jerk
Decreased — absent — normal	2	..
Absent — present — absent	..	1
Moderate change:		
Decreasing	6	9
Increasing	4	6
Absent to present	3	2
Present to absent	3	2
Stationary	16	14
Constantly spastic	11	7
Constantly decreased or absent	10	13

Superficial Reflexes

Plantar	Bilateral	Unilateral
Normal throughout	22	..
Positive throughout	4	1
Negative to positive	4	..
Questionable to negative	..	1
Positive to negative	..	1
Negative to positive to negative	1	..

Abdominal	
Present and normal	31
Absent	2
Absent to present	1

The preceding tabulation cannot give a clear picture of the changing condition of the deep reflexes. In analyzing individual cases, one finds:

Case 1.—The achilles reflex changed from absent to present with an increase in the patellar reflex from —3 to —2.

Case 3.—The patellar reflexes changed from —2 to —3 to —2 to —1, while the achilles reflex remained constantly absent.

Case 4.—The patellar reflex changed from absent to —2, while again the achilles reflex remained absent.

Case 9.—The patellar reflex returned from absent to —3. The achilles reflex was absent throughout. The plantar reflex changed from a unilateral dorsal flexion to plantar flexion.

CASE 15.—The patellar reflex varied from normal to —2 to + 3, while the achilles showed a parallel change of —1 to —2 to normal.

CASE 19.—There was a unilateral return of the achilles reflex from absent to normal.

CASE 22.—The patellar and achilles reflexes were absent, and the plantar reflexes were normal on two examinations. Seventeen months later, the patellar and achilles reflexes were all + 3, and the plantars showed strongly positive Babinski signs.

CASE 23.—A marked variability in the patellar reflex was shown by changes from —2 to + 1 in eighteen months, and to a total absence six months later. The achilles reflex varied from absent to + 4 with clonus and back to absent again. The plantar reflexes showed a parallel change of normal, positive Babinski sign, normal.

CASE 27.—There were changes in the achilles reflex of + 2, —2, normal.

Twelve patients showed decreasing or absent patellar reflexes; whereas twenty showed decreasing or absent achilles reflexes. From this it would seem that the greatest variability was exhibited by the knee jerks. However, one finds the achilles reflex returning in three cases, in one to disappear again. A continued observation will probably be necessary to determine the true tendency, which at present seems to be a gradual decrease to a permanent loss of the achilles reflex.

On comparing the symptoms of the posterior column with those of the lateral column, one is impressed by their constancy and progressiveness. Four patients remained normal over periods of seven, sixteen, thirty-five and fifty-four months, respectively. Eighteen patients showed a progressive decrease, and in eleven patients the sensation was absent at first, and remained so. One patient (case 6) gave evidence of returning vibratory sensation after an interval of one year and one month, although the deep reflexes had increased somewhat.

Paresthesias usually disappear soon after treatment is instituted and remain absent as long as the blood is kept at a high standard, but here also there is some variability. In case 15, the patient had complained of generalized body tingling and coldness of the outer aspects of both forearms and legs. This has shown a change from time to time, but has returned in spite of a high hemoglobin and red cell count.

Improvement in disturbance of gait has been at times miraculous. It is not unusual to see a helpless patient regain ability to walk unaided by crutch or cane. In case 25, the patient entered the hospital unable to control the lower extremities in any degree. At the end of three years he has progressed from crutches to one cane, and soon he will discard that. Reflexes and deep sensation have not changed, and he is unable to walk in the dark. Sphincter control has not changed, there being occasional rectal and vesical incontinence. The patient in case 3, when first seen in 1926, could walk with difficulty and could not drive a car. Improvement was rapid, and in 1928 she was driving a car again,

although here also the patient retained a positive Romberg sign and unchanged reflex and sensory alterations. The patient in case 22 was unable to walk without assistance when he entered the hospital. He walked well when discharged, but in the two-year interval between examinations he developed a spastic gait and required a cane for assistance. In case 33, the patient was unable to walk when first examined, but at the end of three months was able to go about unaided and at the end of four years walked with little evidence of impairment. Another patient, case 34, also showed a marked improvement in gait.

Improvement in gait in these cases cannot be interpreted as the result of anything but improved strength, since in every case the reflex and sensory changes have remained essentially the same.

Other cases have shown no change in an already impaired gait (cases 4, 17, 23 and 29), while still others have developed disturbances in gait while using liver in adequate amounts (cases 11, 12, 14, 15, 18, 20, 21 and 32). A physician's father, case 18, who has been constantly maintained at a high hemoglobin standard, has shown a steady progress in all symptoms and now cannot walk alone. The patient in case 20, while under constant observation and while maintaining a high hemoglobin percentage, developed a spastic gait that prevented her from walking without help.

Changes in coordinate movements occur and generally parallel the changes in gait. Occasionally one finds, as in case 13, an increasing impairment in coordination while the gait remains normal, or, as in case 22, an impairment in coordination that remains constant while the gait varies.

Mental changes, mild or severe, generally show a marked improvement under liver therapy. Marked delusional trends have disappeared (cases 7, 21 and 25), and depressed patients have regained a normal outlook (case 6). The response is in keeping with the general nutritional changes and improved condition of the blood.

Since the introduction of liver therapy in 1926, I have been impressed by the prompt and persistent response of the blood-forming organs to liver and liver extract, and recently to ventriculin. Unfortunately no such constant response has been noted in the neurologic symptoms. Many reports have appeared, some optimistic, some pessimistic. Of the authors reporting improvement, Wilkinson[10] stated: "Several cases with early posterior lateral involvement of the spinal cord have shown remarkable improvement and paresthesias of the hands and feet (without alteration of reflexes) have been almost completely cured in every case."

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10. Wilkinson, J. F.: Treatment of Pernicious Anemia with Hog's Stomach, Brit. M. J. **1**:85 (Jan. 17) 1931.

Richardson [11] stated that "certain nervous system symptoms clear up either entirely or to a greater or lesser degree," but he did not report what symptoms improve. Smith [12] stated that mental and cord changes improved under liver therapy. Ungley and Suzman [13] were perhaps the most enthusiastic of all. They stated emphatically that there are regressive changes in all fields. Starr [14] suggested that keeping the red cell count above 5,000,000 may prevent progressive cord changes, basing his statement on observations in eight cases over a considerable time.

Bubert [15] reported a case in which the gait improved remarkably. The knee reflexes changed from absent to spastic. No mention was made of vibratory sensation.

Opposed to these optimistic views one finds many discouraging reports. Fahr [16] stated that "The toxic changes which are not dependent on the blood picture are not influenced by the liver therapy, and therefore progress is unhindered and causes death. Most important among these is funicular myelitis." Curschmann,[17] McAlpine,[18] Sturgis,[19] Krause [20] and others reported failure of improvement.

Reflex changes, both deep and superficial, changes in gait, disappearance of subjective sensory phenomena and improvement in mental status undoubtedly occur; but to state that these changes are the result of liver therapy and to regard loss of paresthesias, changes in pyramidal tract signs and improvement in gait as evidence of regression of pathologic changes in the cord are, I believe, erroneous.

To state that the maintenance of a high hemoglobin content and a red count of 5,000,000 will prevent or improve cord changes is answered by

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11. Richardson, Wyman: Pernicious Anemia, New England J. Med. **200**:540 (March 14) 1929.

12. Smith, L. H.: Mental and Neurologic Changes in Pernicious Anemia, Arch. Neurol. & Psychiat. **22**:551 (Sept.) 1929.

13. Ungley, C. C., and Suzman, M. M.: Subacute Combined Degeneration of the Spinal Cord: Effects of Liver Therapy on the Nervous Phenomena, New Castle M. J. **9**:67 (Jan.) 1929.

14. Starr, Paul: The Prevention of Spinal Cord Degeneration in Pernicious Anemia, J. A. M. A. **96**:1219 (April 11) 1931.

15. Bubert, H. M.: Subacute Combined Sclerosis, J. A. M. A. **90**:903 (March 24) 1928.

16. Fahr, T.: Liver Therapy in Pernicious Anemia, Deutsche med. Wchnschr. **57**:8, 1931; abstr., J. A. M. A. **96**:1186 (April 4) 1931.

17. Curschmann, H.: Failure of Liver Therapy to Influence Nervous Disorders, Med. Klin. **25**:1767 (Nov.) 1929.

18. McAlpine, Douglas: Review of the Nervous and Mental Aspects of Pernicious Anemia, Lancet **2**:643 (Sept. 28) 1929.

19. Sturgis, C. C.; Isaacs, Raphael, and Riddle, M. C.: Treatment of Pernicious Anemia by Liver Feeding, Surg., Gynec. & Obst. **50**:234 (Jan.) 1930.

20. Krause, F.: Failure of Liver Therapy in Nervous Manifestations of Pernicious Anemia, Klin. Wchnschr. **8**:2177 (Nov. 19) 1929.

cases such as 18 and 20, in which the disturbance in gait developed while the patient was under constant careful observation. Such a hopeful outlook is made more doubtful by a patient examined since this paper was begun.

A woman, aged 50, was referred as having a case of functional disorder. She had been examined a number of times, and nothing significant had been found, except achlorhydria, which she said had existed for ten years. The family history revealed that the father had died of pernicious anemia. She was married, but there had been no pregnancies. Her past health had been excellent. The history of the present illness included tingling and numbness in the fingers for nine months, and in the lower extremities for three months, progressing to the middle of the thighs. She complained of being awkward and of bumping into things. Fatigue was marked, and mental depression had developed during the past month. Examinations of the blood on several occasions during the past year had shown no abnormalities. Physical examination gave negative results. A neurologic examination revealed: cranial nerves normal; coordination impaired in the heel-to-knee test; biceps reflexes normal; patellar reflex normal on the left and —2 on the right; achilles reflex —3 on the left and —2 on the right. The abdominal reflexes were absent and the plantar reflexes normal; sensation was undisturbed, except for quantitative vibratory sensation, which was absent over both first toes and both internal malleoli, and was felt for four seconds over the right and five seconds over the left tibia; on the right anterior sacral spine, vibratory sensation was felt for five seconds and on the left for nine seconds. The blood at this time showed: hemoglobin, 80 per cent; red cells, 4,480,000, with anisocytosis, 2 + and occasional poikilocytosis; white cells, 7,650; platelets, 160,000. The Wassermann and Kahn reactions were negative. Ten days later, after liver feeding, the hemoglobin was 100 per cent; the red count, 4,490,000; the platelet count, 220,000, and the white count, 8,450. There had been no improvement in the paresthesias.

If the maintenance of a high hemoglobin content will prevent cord symptoms from developing, one would not expect this patient to present such definite evidence of posterolateral sclerosis before anemia was present.

Reversing the argument, one sees examples of what might be considered improvement in patients whose hemoglobin has never attained a height above 80 per cent, as in case 6, the only one in the series to show any return of vibratory sensation.

What happened to the neurologic signs and symptoms before liver therapy was introduced? It is known that the paresthesias (which I do not regard as originating in pathologic changes in the cord, but which are so regarded by many) appeared and disappeared, and that improvement in gait was not unknown. As to the signs in the pyramidal tract and the posterior column, I can find no report of repeated examinations, without which there is no way of knowing whether variability in the observations is the usual occurrence. This is unfortunate because one does not dare to deny a patient the privilege of liver therapy to answer the question. I cannot feel, therefore, that one is justified in claiming that the variation

in the signs of involvement of the pyramidal tract as noted in this series is the result of liver therapy.

The differences of opinion concerning the effect of liver on the combined degeneration of the spinal cord result, I believe, from observation over too short a time, and possibly from misinterpretation of the changes seen in the patient. I believe that a definite knowledge can be obtained only after larger series of cases are studied by individual observers over longer periods.

At present my impression is that liver therapy has no effect on the pathology of the spinal cord once it is established, and that it is questionable as to whether it has any effect in preventing pathologic changes from developing.

SUMMARY

Of 189 cases of proved primary anemia, 97.77 per cent showed evidence of combined degeneration of the spinal cord as evidenced by posterior and lateral column signs. Paresthesias and subjective complaints are not used in obtaining this figure.

The symptoms of cord involvement may vary from a quantitative loss of vibratory sensation over the first toes to fulminating involvement leading to an early death.

The earliest conclusive evidence of involvement of the spinal cord is a quantitative disturbance of the vibratory sensation over the distal portions of the lower extremities. Disturbance of vibratory sensation in the upper extremities is rare.

A careful quantitative study of vibratory sensation aids in diagnosis when other symptoms, including blood changes, would not suggest a diagnosis of pernicious anemia.

Deep reflex changes occur slightly more often than involvement of the posterior column (90.4 per cent as against 89.3 per cent), but without signs of pathologic changes in the posterior column they do not suggest a diagnosis of pernicious anemia.

All cases of pernicious anemia probably would show evidence of combined degeneration of the spinal cord at some time during the progress of the disease if examined as a routine at stated intervals.

Considerable confusion has grown out of conflicting claims for and against the effectiveness of liver therapy in controlling cord symptoms.

Thirty-four cases have been observed over a considerable time, and repeated neurologic examinations have been made.

Analysis of the findings at these repeated examinations emphasizes: 1. Lateral column signs and symptoms show marked and unaccountable variations toward a persistent loss of the achilles reflex in some, or toward a persistent spasticity in others, indicating a preponderance of involvement of one part of the cord or the other. 2. There is an early,

progressive, permanent disturbance and finally a loss of the vibratory sensation, first in the distal portions of the lower extremities and later progressing upward, but seldom involving the upper extremities.

The early and persistent use of liver cannot be credited with causing reflex changes, unless it can be demonstrated that such changes are not part of the usual picture and did not occur before liver therapy was introduced.

Liver therapy has no demonstrable effect on posterior column symptoms.

Improvement in coordinate movements, station and gait is to be attributed to improved muscle tone, strength and muscle training, since no parallel change occurs in the signs of cord involvement.

Improvement in mental condition has been noted, and psychotic trends have been reduced in degree and in proportion to an improvement in the general condition.

Paresthesias usually disappear, but may persist or return.

Maintenance of a high hemoglobin content and red cell count should be attempted, but cord changes have occurred in spite of the condition of the blood.

CONCLUSIONS

A high percentage of cases of primary anemia show evidence of subacute combined degeneration of the spinal cord, and if carefully examined, all cases would probably show cord changes at some time during the course of the disease.

Because of the diversity of my observations in this series of cases, I do not choose to draw conclusions as to the effect of liver therapy on combined degeneration of the spinal cord. I do not believe that conclusive evidence can be presented until a larger series of cases has been examined and reexamined by one observer over a considerable period of time.

My impression, however, at present is that liver therapy has little or no effect on the pathology of the central nervous system.

ABSTRACT OF DISCUSSION

DR. O. H. PERRY PEPPER, Philadelphia: Dr. Ahrens is quite properly cautious in drawing conclusions as to the efficacy of liver therapy on the posterolateral sclerosis of addisonian anemia. That the evidence concerning the effect of liver therapy must be inconclusive is obvious when one realizes that for more than twenty-five years the literature has discussed the variability of reflexes and the influence of the patient's general condition on the cord symptoms in cases in which the patients were not treated by liver. Most writers of recent articles, as, for example, Smithburn and Zerfas,[21] are equally conservative. Certainly my own views coincide.

21. Smithburn, K. C., and Zerfas, L. G.: The Neural Symptoms and Signs in Pernicious Anemia, Arch. Neurol. & Psychiat. **25**:1100 (May) 1931.

If the reflexes vary without treatment, how can we draw conclusions about the effect of liver? That they do vary I had demonstrated to me at a clinic I was giving in Iowa. I was demonstrating a patient with addisonian anemia in whom a hospital record of some months previous stated that the knee reflexes were absent. The patient was sitting near the edge of a raised platform, and as I leaned down to tap the patellar tendon, I was within a foot of the edge of the platform, standing almost in front of the patient. The tremendous and unexpected reflex kick that I elicited came within an inch of toppling me off the platform.

It is far easier to believe that liver therapy acts only through its effect on the patient's general strength and spirits than that it has any specific beneficial action on the cord changes. One needs only to visualize the sclerotic and degenerative character of the changes in the cord of primary anemia to be persuaded that true improvement of cord function would come only from actual regeneration of fibers, a happening that probably does not occur.

Similarly, we must be cautious in accepting the reports of benefit by liver therapy in disseminated sclerosis as recently claimed by Goodall and Slater.[22]

In spontaneous remission, as well as after liver therapy, there will often be marked relief from paresthesias, psychoses and weakness. With the latter will come improved control of the extremities, an improved gait, etc.

I have recently had an instructive patient who has had symptoms justifying the diagnosis of posterolateral sclerosis of the anemic type for over fifteen years and who has been treated with adequate amounts of liver for several years under my care. In spite of this, her posterolateral sclerosis has become worse, and recently she became anemic. An increase in the specific treatment produced no reticulocyte crisis, nor any improvement. The administration of iron, however, promptly raised the blood count, and with this improvement in blood count occurred a simultaneous improvement in her general condition, which, carelessly interpreted, seemed to include actual improvement in the spinal cord symptoms. In this case, it seems to be clear that the benefit was wholly due to the improved general condition, a result brought about, not by specific substance, but simply by iron.

Dr. Ahrens properly warns us of the difficulty of judging the results of our therapy. I would like to warn you that it is easy to mistake for primary pernicious anemia certain forms of secondary anemia; for example, the anemia due to carcinoma of the colon and that form of dietetic anemia called chronic chlorosis.

For a clearer understanding of this subject of posterolateral sclerosis of the anemic type, the unknown cause of the cord degeneration must be discovered. As far as I know, this is unknown; it is not enough to attribute it to some hypothetic toxin, nor does it seem to have a common cause with the anemia. To me it seems more likely to be due to focal tissue anemia perhaps the result of a failure of the abnormally large red cells to pass through the finer capillaries of the cord in sufficient numbers to maintain an adequate oxygen supply to the readily injured tissues of the central nervous system.

Dr. James Ayer, Boston: Dr. Woltmann asked me in Dr. Minot's absence if I would get his data and present it at this meeting. I called Dr. Minot and found that he had already started for the meeting, so I have not been able to get the data.

There is one point I shall speak of beside the point Dr. Pepper stressed. Drs. Minot and Castle and their co-workers insist, as I think others insist, that in refractory cases (and practically all of the cases of neurologic primary anemia

22. Goodall, A., and Slater, J. K.: Brit. M. J. **1**:789 (May 9) 1931.

are refractory) we should not be satisfied with a preliminary gain, but we should push the liver beyond the point that seems necessary, certainly beyond the point that will give relatively normal blood; that the blood should be made as near perfect as possible before we let up in any way, and that a high level should be maintained. In other words, do not stop with an early gain either in blood or in nerve symptom improvement, but push the liver.

Also, they feel strongly that liver itself, purée of liver, is worth a great deal more, at least in neurologic conditions, than the extract.

I have been rather interested in the cases that Dr. Ahrens eliminated from his group, namely, those that have the symptoms of the disease, but insufficient signs on which to base the diganosis of combined system disease. Those cases, I think Dr. Ahrens will agree, have reacted very promptly to liver therapy, whether or not the blood is characteristic.

I have been further interested in two cases of typical combined system disease with symptoms and signs thereof, with blood not characteristic and with hydrochloric acid present, a condition that I am told can not occur, and yet which seems to occur occasionally. One of the patients has done well on liver therapy; the other case is too recent to say.

Dr. Richard S. Ahrens, Minneapolis: I am glad Dr. Pepper emphasized the fact that diagnosis is not always easy in these cases. In this series, however, all of the cases have been carefully studied at the Nicollet Clinic or at the Minneapolis General Hospital, and in cases in which there has been question, blood has been submitted to capable hematologists, and observation over a period of time has been allowed before a definite diagnosis was made.

Dr. Ayer asked what had been done with or about cases that did not show neurologic signs. It has happened that in the series of thirty-four with which I have dealt, all of the patients have shown signs, and at the present time I have none under observation, nor am I in contact with any one with the diagnosis of primary anemia who does not show cord signs. This is, of course, unfortunate.

ATLANTO-OCCIPITAL EVACUATIVE TREPANATION IN CONTUSIONS OF THE BRAIN

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The treatment for severe traumatic injuries of the brain may be divided into two large classes: The first class is surgical and is applied to extensive hemorrhages that can be localized. In this group are included such lesions as hematomas, extradural or subdural, and certain subarachnoidal hemorrhages, the situation of which, in the neighborhood of the motor area, for example, provokes localizing convulsions.

The second is the class of conservative treatment, the principal weapons of which are watching and waiting, and lumbar punctures. The latter is the treatment *par excellence* for pure concussion, that is, for a state of paralytic cerebral vasodilatation that follows traumatisms that are not severe enough to provoke anatomic or histologic cerebral lesions. In such cases, lumbar punctures permit alleviation of the hypertension that results from hypersecretion of cerebrospinal fluid caused by cerebral and pial congestion (the plexus is but pia invaginated in the proportion of 1 sq. mm. of pia to each plexus) and by the reduction of space due to the increased bulk of edematous brain.

Similarly, in traumatic sinus thrombosis, lumbar punctures may constitute the only therapeutic measure, and may relieve the convulsive attacks caused by the hypertension secondary to venous stasis and increase in outflow of the cerebrospinal fluid dialyzed from the surface of the 2 sq. mm. of choroid plexus.

Such intermittent drainage of cerebrospinal fluid is also of therapeutic value in the majority of cerebral contusions with diffuse lesions of the brain and intrameningeal extravasations. In these cases there are several factors that tend to increase greatly the bulk of fluid in the cranial chamber. In addition to the congestive hypersecretion referred to, two other causes must be considered, namely, obstruction of the emunctories of the cerebrospinal fluid by destroyed blood cells in the arachnoid villi and perivascular spaces, and irritation of the serosa by the escaped blood. Therefore, lumbar punctures are particularly indicated and may not only save a life but allow the conservation of a normal function if not an integral anatomic restitution of the brain.

From the Canton Hospital.

However, if the blood that has extravasated into the subarachnoid spaces accumulates in the great cisterns of the base, this form of treatment is not always sufficient. This is because of an important fact which it behooves the surgeon to emphasize: In contusions of the brain the blood shed in the cerebrospinal fluid fissures and channels flows from one subarachnoid space to another, reaching the most dependent part of the skull, that is, the peribulbar region. For example, from the cisterna fossae sylvii the blood can flow into the cisterna interpeduncularis, then into the cisterna pontis and the cisterna magna. Even in contusions of the upper part of the brain, the bleeding may run toward the base of the skull by way of the rolandic or sylvian fissure. Professor Askanazy, chief of the Institute of Pathology of Geneva, has on several occasions demonstrated these observations to me in postmortem examinations in cases of fatal contusion of the brain. In his thesis, Duret, as early as 1878, referred to such lesions, pointing out that they may destroy cranial nerves, producing a symptomatology of value in diagnosis. But there is something of even greater importance. The clots accumulated in mufflike fashion around the bulb may, by compression or choking, perturb the function of the respiratory and circulatory centers or cause a fatal anemia of the medulla by direct mechanical action.

Of course it must be recognized that by far the larger number of deaths from cerebral contusion result from a direct inhibition of the vital centers rather than from an extrinsic damage to the medulla. Though in the majority of cases therapeutic measures are of no avail, there is a small group, until now neglected, that may require operation. This is the group in which, as has already been emphasized, the hemorrhage accumulated at the base of the brain becomes a vital danger, and in which not the primary lesion of the brain but this secondary hemorrhage causes cessation of heart beat or respiration.

The diagnosis of contusion of the brain, far from constituting a contraindication to operation, in my opinion, may become a direct indication for surgical intervention, since the contusion, in itself beyond therapeutic attack, gives rise to complications against which surgical intervention may be effective.

There is another point to be considered. The blood that has oozed from the cerebral or pial lesions to the subarachnoid spaces may gather not only in the peribulbar infundibulum but lower in the cervical region. Here the dural sheath of the cord may be so closely applied that the coagulated blood is checked, and thus a barrier, more or less complete, is established between the cephalic and the spinal fluid. When a lumbar puncture is performed, this block may mask the considerable hypertension which raises havoc in the skull, especially as this procedure, because of the suction exerted, favors the wedging of the

peribulbar clots downward to form a compressing sleeve. The value of lumbar puncture, therefore, may be deceptive in just those cases in which it is recommended as the method of choice.

Evacuation trepanation in the posterior part of the skull, on the other hand, is designed to eliminate the irritative or compressive formations of blood around the medulla and to liberate the brain from a hypertension that may cause definite functional disorder. It is well known that in severe traumatisms treated by trepanation, the posttraumatic condition characterized by inability to work, dizziness, headache, blurring of vision and insomnia is less likely to occur than in cases in which operation is not performed. This postconcussion syndrome, which has been described by Pierre Marie, is influenced to a notable degree by evacuation of the blood resulting from the injury to the brain, thus preventing secondary hypertension. Experience has shown that concussion of the brain with open fracture of the skull is less often followed by late functional cerebral sequelae than when associated with a closed fracture.

INDICATIONS FOR OPERATION

The operation is designed to relieve compression or irritation of the medulla and is indicated whenever a subarachnoidal hemorrhage of unlocalizable origin reaches the bulb and, whatever its origin, causes accidents that lumbar puncture, even when repeated, does not alleviate.

But such a diagnosis of bulbar disturbance is difficult in cases of injuries of the skull because the symptoms of a lesion localized in the bulb are masked by those of a general involvement of the brain. For example, glossolabiolaryngeal palsy, with dysarthria (paralysis of the seventh and twelfth nerves), dysphagia (paralysis of the ninth nerve), deviation of the jaw (paralysis of the fifth nerve), and acceleration of the pulse rate to 120 and even 160, might be of diagnostic significance in the recognition of a bulbar syndrome were these symptoms not also elements of other traumatic cerebral syndromes. Again, stertor may be a sign of palsy of the soft palate due to an inhibition of the tenth nerve, but may also occur in the absence of any bulbar disturbance, being only a manifestation of general abolition of muscle tone characteristic of such a traumatic condition. Similarly, the pharyngeal reflexes may be abolished in the general insensibility resulting from inhibition of the cortical centers. Twitching of the eyelids and of the labial commissures, nystagmus, rapid alternate contractions and dilatations of the pupils and spasmodic contractures of the jaw, may all be signs of bulbopontopeduncular irritation as well as of cortical lesions. Dyspnea, projectile vomiting, marked acceleration or slowing of the pulse and hypertension or hypotension are also not exclusively the result of bulbar disturbance. Beginning papilledema is perhaps of more diagnostic value,

because it is an early symptom of increased intracranial pressure chiefly when due to obstruction of fluid in the posterior fossa. Corneal areflexia is possibly the most significant sign of sensorimotor disturbance of the medulla, not because the arc of this reflex is entirely bulbopontile, but because the corneal reflex is one of the last to disappear in severe traumatism of the brain.

REPORT OF CASES

CASE 1.—A man, aged 32, was found unconscious on the street at 5 a. m. He answered questions, but with difficulty. He could not explain what had happened, but it was reported that he had fallen from a motorcycle. He was immediately sent to the hospital.

Examination.—Several small wounds of the scalp were present. The pulse rate was 82. The patient complained of severe headache which he could not localize. The pupils reacted well; the reflexes were normal, and the Babinski sign was absent. There was no disturbance of sight or hearing and no vomiting. The patient, desiring to go home immediately, was discharged against advice.

Course.—On the following evening, after a relatively quiet day passed at home in bed, the patient had a generalized convulsion. It was stated that he had never before had a similar attack. Examination by a local physician revealed that the neck was stiff, the knee jerks were markedly diminished, the Kernig sign was positive and the Babinski sign negative. The temperature was 38 C. (100.4 F.). The patient was very uncooperative, and continually complained of severe headache. On lumbar puncture, there was an initial pressure of 82 mm., which after escape of 22 cc. of bloody fluid fell to 30 mm.

The patient was again transferred to the clinic. Roentgen examination showed a fracture of the vault, extending to the base. In the afternoon, a second convulsion occurred, which was characterized by generalized tonic and clonic movements. The patient was drowsy, did not remember anything, answered questions poorly, and continued to complain of headache. The temperature was 38.8 C. (102 F.). The pulse rate, which had been 140 during the convulsion, continued to be rapid (125). Examination of the eyes showed mydriasis and blurring of the papilla without measurable elevation. The knee jerks were obtained with difficulty.

The findings may be summarized as follows: (1) a long interval between the accident and the appearance of the first manifestations of cortical irritation; (2) two convulsive attacks without premonitory or localizing signs; (3) the presence of bloody cerebrospinal fluid on lumbar puncture; (4) a progressively accelerated pulse; (5) markedly diminished knee jerks; (6) the absence of sensory and motor disturbances; (7) rapidly increasing drowsiness.

The case seemed to be one of cerebral contusion, with subarachnoid hemorrhage. The progressive alarming evolution of the symptoms indicated operative intervention. The absence of any localizing signs, however, prevented operation at the site of the lesions, which apparently were generalized.

Operation.—As there were no localizing signs and the cerebrospinal fluid was bloody, a posterior atlanto-occipital trepanation was decided on. With a ventral decubitus, under ether anesthesia, a median posterior occipitocervical incision was made, with liberation of the posterior arch of the atlas, which was resected with rongeurs. Hemorrhage was profuse owing to extreme congestion of tissues. A paramesial incision in the atlanto-occipital membrane was made. Under great pressure, a large amount of highly hemorrhagic fluid (about 300 cc.) escaped. The

fluid that gushed out contained numerous clots, and in order to facilitate their evacuation, a larger opening was made in the atlanto-occipital membrane. During this decompression the pulse rate dropped from 128 to 96, and respiration became obviously easier and deeper. A drain was left in the opening and the wound was closed in layers.

Postoperative Course.—On the following day the patient was no longer drowsy and responded promptly to questions. The headache had greatly diminished. The knee jerks were active. The temperature dropped to 37.8 C. (100.2 F.) in the morning and 37.2 C. (98.8 F.) in the evening. The fundi were less congested. The amount of bloody cerebrospinal fluid evacuated during the day was about 1.5 liters, necessitating repeated dressings.

On the second day there was a notable improvement in the condition. The headaches had almost entirely disappeared. The cerebrospinal fluid, although not diminished in flow, contained less blood.

Five days later, the fluid was clear and greatly diminished in amount. There were no more headaches. The knee jerks were active, the Babinski sign was negative and the achilles reflex was normal. There were peri-ocular ecchymoses on the right side.

On the tenth day, the drain was removed. Three weeks later, the patient was up and about, and twenty-eight days after the operation he was discharged in a good condition. A few weeks later, he returned to work as a chauffeur. He reported for examination five months afterward, at which time he was entirely free from symptoms.

Comment.—In this case the operative finding of an amazingly large amount of cerebrospinal fluid under great pressure and mixed with an abundance of blood clots, as well as the favorable effect of posterior drainage on the function of the medulla, fully justified the attempt to relieve the bulb. Following the evacuative trepanation, the respirations became quieter and the pulse returned to normal. It may be of interest at this point to refer to the figures taken from the temperature chart. It is interesting also to note that the pulse rate, which was 124 at the beginning of the operation, fell to 96 at the end of the evacuation and to 88 a day later.

Time	Pulse	Temperature
6 p. m. (shortly after operation)	106	38.1 C. (100.6 F.)
7 p. m.	108	
9 p. m.	100	
Midnight	96	
4 a. m.	100	
6 a. m.	92	37.8 C. (100 F.)
10 a. m.	96	
Noon	92	
6 p. m.	88	37.2 C. (99 F.)

Although following lumbar puncture there was a rapid fall in intraspinal pressure, a marked hypertension of the fluid persisted in the cranial subarachnoid space. This is attributed to the fact that the communication between the cranial chamber and the spinal canal was

obstructed, probably by clots. The value of decompressive lumbar puncture, as has been pointed out, is not absolute, and in this case failed to be a sufficient therapeutic measure. The sudden decompression of the brain apparently had no ill effect.

CASE 2.—A man, aged 43, was admitted to the hospital in the evening in coma. He had lost blood from both nostrils and from the left ear. The temperature was 36.8 C. (98.4 F.) and the pulse rate 88. A slight ptosis of the left lid was noted. The pupils reacted sluggishly. The knee jerks were active; the Babinski sign was negative, and there was no ankle clonus. The cremasteric reflexes were normal. On lumbar puncture, only a few drops of almost pure blood, not under increased pressure, were obtained.

During the night the patient became very agitated. At times he was lucid, and at other times he was in a state of semisomnolence from which it was difficult to arouse him. In the early morning, attacks of vomiting occurred. Bilateral mydriasis was present. The abdomen was tense, and the abdominal reflexes were abolished. The tendon reflexes were hyperactive; the Babinski sign was negative; the cremasteric reflex was abolished on the right, and there was ankle clonus on the right. Thinking that there must have been an error in the technic of the lumbar puncture, I repeated this procedure, and obtained a few cubic centimeters of highly bloody fluid, under a pressure of 30, which dropped immediately. The temperature now was 37.2 C. (98.8 F.), and the pulse rate 118. The respirations were shallow, irregular and exclusively thoracic, at the rate of 42 a minute. At frequent intervals the patient strove to take a deep breath, as though gasping for air. He continued to be exceedingly agitated, and complained of severe headache.

The impression was that the condition was contusion of the brain with subarachnoid hemorrhage and probable compression of the medulla.

Operation.—As the patient's condition grew progressively worse it was decided to intervene. At noon, under ether anesthesia, a posterior median cervical incision from the epiphysis of the fourth cervical to the occipital protuberance was made, with liberation of the posterior arch of the atlas and resection with rongeurs. The atlanto-occipital membrane was then incised on a paramesial line. Immediately, the fluid gushed out under marked pressure. It was mixed with blood clots, and about 400 cc. was evacuated. The pulse rate, which was carefully observed during the operation, dropped from 118 to 72. The respirations, which until then had been labored in spite of the anesthesia, became quieter. The pulse rate remained at from 72 to 78 until the operation was completed. As soon as the fluid ceased to flow, the wound was closed in layers, with a drain fixed in its lower part.

Postoperative Course.—The pulse rate varied from 88 to 92, 72, 84 and 82 until midnight. The temperature at 4 p. m. was 37.2 C. (98.8 F.). During the night, the pulse gradually became accelerated to 100, 104, 108, 132, 130 and 134 toward early morning. It was then noted that the dressing was not wet through as is usual in such cases. On examination, it was found that drainage was not effective. The edges of the wound were therefore spread apart, and a large quantity of fluid under pressure escaped. A longer drain was then placed in the wound. The patient, who had had a severe headache, was immediately relieved and declared that he could breathe more easily. The pulse rate dropped from 132 to 75. In spite of the fact that he had been in an agitated condition for several hours, he soon became quiet.

Following this it was necessary to change the dressing, which although very thick, had become wet through. The pillows and the mattress were soaked by hemorrhagic fluid.

A day later, the amelioration was still more marked. The patient was in a lucid state. The fluid continued to run freely and abundantly, but was no longer as bloody as previously. The temperature in the evening was 38.2 C. (100.6 F.), and the pulse rate was 100, now corresponding with the temperature. There were no more headaches. The knee jerks were normal, but the abdominal reflexes were absent.

Two days later, the patient was absolutely clear mentally. The amnesia had disappeared, and he was able for the first time to tell in detail the story of the accident. There was a decrease in the amount of escaping fluid, which was now only faintly sanguineous. The knee jerks were normal, but the abdominal reflexes were still absent; the cremasteric reflexes were present.

Four days later, the fluid was clear, and escaped only when the head was in certain positions. The drain was removed.

On the twelfth day, only a slight amount of fluid flowed from the lower end of the operative incision.

On the eighteenth day, the patient was up and about. The wound was dry. The abdominal reflexes were now obtained for the first time, although weakly. All the other reflexes were normal.

At the end of the third week, he was discharged in an excellent condition.

Subsequent Course.—A year later, the patient reported for a demonstration before the medical society of Geneva, at which time he was absolutely free from symptoms, having returned to work as a tradesman soon after his recovery.

Comment.—In this case again, following the evacuation of a tremendous amount of hemorrhagic fluid by atlanto-occipital trepanation, there was an immediate improvement in the patient's condition. The recurrence of the clinical symptoms that developed when the drain was temporarily obstructed demonstrates the desirability of continued drainage for effective therapeutic action on the medulla.

It illustrates, moreover, the futility of lumbar puncture in certain cases of cerebral hypertension, even when considerable amounts of fluid are removed. It is clear that a suboccipital puncture, though repeated a number of times, could not be a sufficient means of decompression. The fact that a peribulbar clot may prevent the obtaining of fluid through the needle gives rise to the added danger of too deep penetration and consequent injury to the bulb.

In neither of the cases reported was a foraminal herniation of the cerebellar tonsils encountered. This can be explained by the fact that the blood extravasated in the subarachnoid space does not form a mass like an extradural or subdural hematoma, but increases the general volume of fluid. The pressure is exerted in all directions. As the peribulbar pressure is the same as that in the anterior part of the skull, there is nothing to produce dislocation of the brain. Nevertheless, the possibility of foraminal herniation must be taken into account.

In conclusion, it may be said that one is led to the presumptive diagnosis of severe disturbance of the bulb by the alarming aggravation of symptoms and the presence of blood in the subarachnoid spaces rather than by a clearcut traumatic bulbar syndrome. The operative

indications may therefore be difficult to determine. Furthermore, as the number of cases in which peribulbar hemorrhage causes death is rather limited in contrast to those in which the fatality is due to direct paralysis of the vital centers, it would be presumptuous to expect successful results in every case of severe contusion of the brain.

ADVANTAGES OF ATLANTO-OCCIPITAL TREPANATION

1. This operation renders aid not only at the site of the primary lesions, but where it is most essential to give relief, that is, to the medulla, whose centers are imperiled by peribulbar clots.

2. It allows the drainage of blood extravasated in the subarachnoid spaces even when the primary lesions cannot be exactly localized.

3. This method of drainage is more effective than the classic temporal procedure: It is made at the most dependent point and from a large lake, whereas the latter is from a region where no physiologic accumulation of fluid exists, and where the blood can exude only by infiltration.

4. In atlanto-occipital trepanation, postoperative hernia of the brain does not occur as in temporal drainage. If a foraminal herniation of the cerebellum should result from increased intracranial pressure, resection of the posterior arch of the atlas would free the strangulated cerebellar tonsils after evacuation of the fluid.

5. The integrity of the skull is preserved.

6. This operation is simple, and necessitates no special instruments such as a trepan, a trephine or a burr.

CONCLUSION

This operation, which to the author's knowledge has not been previously described as a therapeutic measure in traumatic lesions of the brain, is proposed on the basis of excellent results in two cases in which it is permissible to conclude, in view of the operative findings, that without such surgical procedure death would have occurred.

BASAL METABOLIC RATE IN EPILEPSY

REPORT OF THREE HUNDRED AND FIFTY CASES

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Endocrinopathies are being mentioned more and more in the literature as possible etiologic factors in convulsive disorders. One of the simplest tests for an endocrine dysfunction is measurement of the amount of oxygen that a person consumes or determination of the basal metabolic rate. The study of basal metabolism is considered by many to be only a test of thyroid activity. However, evidence is fast accumulating to show that the basal metabolic rate is also influenced by disturbances of the other glands of internal secretion, such as the gonads, the suprarenals and the pituitary.

Comparatively few reports have been made regarding the basal metabolism in epilepsy, and they are at such wide variance that one is inclined to discount the suggestion that there is any constant change in this disease.

In 1922, Boothby and Sandiford,[1] working at the Mayo Clinic with 22 epileptic patients, found that the rate in 23 per cent was below minus 10 per cent, and in a subsequent report 24 more cases were studied with 21 per cent minus readings and 16 per cent of readings above normal. Bowman and Fry,[2] 1925, in 15 patients found 12 with minus readings and 3 with plus readings. Nielson,[3] in 1925, in a study of 18 cases concluded that there is apparently a slight but definite decrease in basal metabolism in epilepsy. Lennox and Wright,[4] in 1928, in 130 patients found that in 23 per cent the basal metabolic rate was below normal limits of minus 10 per cent and that the average for the entire group was 3 per cent below normal. Lennox and Cobb[5] in their classic

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From the Department of Pathology of Craig Colony.

1. Boothby, W. M., and Sandiford, I.: Summary of the Basal Metabolism Data on 8,614 Subjects with Special Reference to the Normal Standards for Determination of the Basal Metabolic Rate, J. Biol. Chem. **54**:783 (Dec.) 1922.

2. Bowman, K. M., and Fry, C. C.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **14**:819 (Dec.) 1925.

3. Nielson, J. M.: Etiology of Epileptiform Convulsions, M. J. & Rec. (supp.) **122**:338 (Sept. 16) 1925.

4. Lennox, W. G., and Wright, L. H.: Studies of the Metabolism of Epilepsy. VII. The Basal Metabolism, Arch. Neurol. & Psychiat. **20**:764 (Oct.) 1928.

5. Lennox, W. G., and Cobb, S.: Epilepsy, Baltimore, Williams & Wilkins, 1928.

monograph on epilepsy stated: "One may say that the majority of adult patients with convulsions, have either a normal or a reduced rate of oxygen consumption."

Schon,[6] in 1930, found 36 abnormal rates, in 130 patients tested, 28 of which were below normal. Against these results are the reports of Talbot, Hendry and Moriarty, in 1924,[7] who found the rate increased in 9 of 11 children tested. Frisch,[8] in 1927, found increased rates in the majority of 40 cases. Notkin,[9] in 1930, in 50 cases, also found that a relatively smaller group of patients had a rate below minus 10 than was shown in the reports of some other workers.

During the past year, I have made 350 basal metabolism tests of epileptics at the Craig Colony, the apparatus used being of the Benedict-Roth type and the computations made from the DuBois scale as modified by Boothby and Sandiford. A standard method of procedure has been adhered to strictly throughout, thereby eliminating any variation in

TABLE 1.—*Metabolic Ratings in Epileptic Patients*

	Cases	Abnormally Low	Normal		Abnormally High
			−10	+10	
Men	150	43	51	31	25
Women	150	54	47	29	20
Total	300	97	98	60	55

results that might arise from varied technic. It should be emphasized that practically all errors of technic and lack of cooperation by the patient give increased readings. As a further check on instruments and methods, 25 nurses of varying ages were given the test.

In the largest group, 150 men and 150 women were tested. No effort was made to select the patients other than that they were required to have sufficient mentality to give proper cooperation. Considering a rate anywhere between minus 10 and plus 10 as normal, only 158, or 52 per cent, came within this range, while 142, or 48 per cent, were distinctly abnormal, 97 being below and 45 above normal. In other words, about half of the cases in this series showed abnormal rates, the number of abnormal minus readings being about twice the abnormal plus readings, as shown in table 1.

6. Schon, H. I.: Endocrionologic Investigations in Epileptics, Acta psychiat. et neurol. **5**:227, 1930.

7. Talbot, F. B.: Hendry, M., and Moriarty, M.: Basal Metabolism of Children with Idiopathic Epilepsy, Am. J. Dis. Child. **28**:419 (Oct.) 1924.

8. Frisch, F.: Der respiratorische Gaswechsel der Epilepiker, Ztschr. f. d. ges. exper. Med. **56**:118 (June) 1927.

9. Notkin, J.: Basal Metabolic Rate in Untreated and Treated Patients with Epilepsy, Arch. Neurol. & Psychiat. **24**:1231 (Dec.) 1930.

As to the sex distribution, very little difference was noted, 68 men and 74 women having abnormal rates, with possibly a tendency for the women to have more minus readings than the men, 54 women having rates below minus 10 per cent as against 43 men.

In table 2 the percentages of cases are arranged according to ages. There is an increase in rate at the time of puberty, followed by a gradual diminution in the percentage of cases showing abnormally high rates, while the percentage of low rates remains fairly constant.

TABLE 2.—*Metabolic Ratings in Relation to Age*

Age	Abnormally Low, per Cent	Normal, per Cent	Abnormally High, per Cent	Total Cases
10 to 15	30	46	24	24
15 to 20	22	40	29	48
20 to 25	26	52	22	47
25 to 30	22	64	14	49
30 to 35	39	47	14	39
35 to 40	38	44	18	17
40 to 45	20	64	16	25
45 to 50	45	55		19
50 to 55	45	55		15
55 to 60	37	63		13
60 to 70	25	75		4

TABLE 3.—*Metabolic Ratings in Relation to the Duration of the Epilepsy*

Duration in Years	Below −10, per Cent	Normal		Above +10, per Cent	Total Cases
		−10, per Cent	+10, per Cent		
1 to 5	22	37	24	17	31
6 to 10	34	29	22	15	58
11 to 15	18	42	20	20	52
16 to 20	37	36	12	15	54
21 to 25	33	33	9	25	30
26 to 30	21	26	32	21	29
31 to 35	48	28	19	5	16
36 to 40	28	14	44	14	16
41 to 45	36	36	16	12	15
46 to 50	33	33	16	16	8

In order to determine whether or not the duration of the epilepsy was a factor in this seeming lowering of the rate in older patients, the basal rate was checked against the number of years elapsing since the onset of the disease. The results are shown in table 3. Nothing of significance can be noted, approximately the same percentages being observed in cases of recent onset as in those extending over a longer period.

Naturally, one would expect to find some difference in the metabolic rates of patients having frequent attacks and of those having only 1 a month. To determine this variation, if any, the patients were placed in 5 groups. Those having 5 or less seizures per month, those having from

5 to 10, from 10 to 15, 15 to 20, and all having more than 20 per month, as shown in table 4. It will be noted at once that as the seizures increase in frequency the percentage of low rates increases and correspondingly the percentage of high rates decreases, while the normal ratio remains constant. In the group of patients having more than 20 seizures per month, 84 per cent gave a minus reading. Whether or not this low metabolic rate is an effect or a cause of the seizures is a problem for speculation and one worthy of considerable research.

TABLE 4.—*Metabolic Ratings in Relation to the Frequency of Seizures*

Average Monthly Seizures	Abnormally Low, per Cent	Normal		Abnormally High, per Cent	Total Cases
		−10, per Cent	+10, per Cent		
1 to 5	22	25	23	20	185
5 to 10	28	31	25	16	66
10 to 15	32	40	18	10	22
15 to 20	35	47	10	8	6
Over 20	37	47	11	5	21

TABLE 5.—*Metabolic Ratings in Relation to Phenobarbital Therapy*

	Abnormally Low, per Cent	Normal		Abnormally High, per Cent
		−10, per Cent	+10, per Cent	
With phenobarbital (158 cases)				
Male	32	38	16	14
Female	30	30	21	19
Without phenobarbital (142 cases)				
Male	26	37	19	18
Female	34	30	26	10

TABLE 6.—*Relation of Metabolic Ratings to the Presence or Absence of Seizures*

	Abnormally Low, per Cent	Normal		Abnormally High, per Cent	Total Cases
		−10, per Cent	+10, per Cent		
Having seizures	32	33	20	15	300
Free from seizures	24	36	18	22	50
Nonepileptic	8	52	36	4	25

Inasmuch as 158 of the 300 patients were receiving phenobarbital at the time of performing the test, it was thought worth while to check these against those who were not receiving such medication. As far as can be ascertained from the data given in table 5 there were as many abnormally high and low rates in the patients taking phenobarbital as in those who were not being treated by this drug .

Near the end of the experiment it was discovered that several patients had been included who had been free from seizures for a period of one

year or more, and so it was thought advisable to establish a separate group of such patients in order to ascertain whether or not the basal metabolic rates differed from those in patients subject to attacks. After careful search, 50 patients were found who could be placed in the seizure-free group. Twenty-seven of these patients had rates within normal limits and 23 rates were abnormal, 12 being below minus 10 and 11 above plus 10. From table 5 it will be seen that although the percentage of abnormal rates is the same in both groups, there seems to be some shifting from the low to the high side in the seizure-free group, which indicates that at least some of the patients may have had an endocrine adjustment that accounts for the period of remission from seizures.

Although the number of nonepileptic controls was small, I think that the results obtained are sufficient evidence that no gross errors in technic were present. Of the 25 nurses tested, 22, or 88 per cent, had rates within the normal limits. They are included in table 6 in order that they may be more readily compared with the epileptic groups.

CONCLUSIONS

1. Fifty per cent of 300 epileptic patients tested showed abnormal metabolic rates; the chances are 2:1 that the rates will be abnormally low rather than high.

2. More females than males show low rates.

3. Age does not seem to be a factor in determining the occurrence of an abnormal rate.

4. The percentage of abnormal rates in patients given phenobarbital does not differ from that in the untreated group.

5. In a group of 50 patients having a remission of seizures over a period of one year or more, the percentage of low rates was not as great as in the group having seizures.

6. The duration of the epilepsy does not seem to be a determining factor.

7. The percentage of low metabolic rates is greater in patients having frequent attacks.

8. Determinations of basal metabolism in epilepsy are of value in offering clues as to the etiology and suggestions for treatment in selected cases.

EXPERIENCES IN THE TREATMENT OF MULTIPLE SCLEROSIS WITH QUININE HYDROCHLORIDE

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Certain observations recently made in the treatment of multiple sclerosis with quinine hydrochloride form the basis of this report. Although several years will be required to appraise the merits of this form of therapy, some of the results already obtained from it suggest that it may be of value. It is true that the evaluation of all therapeutic results in multiple sclerosis is seriously complicated by the nature of the disease itself. Chief among these complications are those incident to the spontaneous remissions, as well as the progressive advance of many symptoms to a point beyond relief by therapeutic measures.

Some recent experimental evidence gave indications that the lesions in multiple sclerosis may be due to abnormal lipolytic activity of the blood on the myelin in the nervous system. These experiments [1] were based on the degree of myelinolysis which resulted from the immersion of the spinal cords of rats in blood plasma from patients with multiple sclerosis as well as from normal persons. Plasma from patients with multiple sclerosis produced more advanced myelinolysis than did that from normal persons. It was concluded, therefore, that the blood in multiple sclerosis can cause decomposition of myelin, and further postulated that such decomposition is due to abnormal lipolytic activity, which, in turn, may depend on the presence of an abnormal lipase. Additional evidence of this abnormal activity has been found in chemical studies, in which the action of serum from patients with multiple sclerosis on lecithin differed from that of normal serum.[2] Similarly, Crandall and Cherry [3] have demonstrated such abnormal activity,

From the Neurological Institute and the Neurological Department of Columbia University.

Read at a meeting of the Neurological Section of the Medical Society of the State of New York, June 3, 1930.

1. Brickner, R. M.: Studies on the Pathogenesis of Multiple Sclerosis, Arch. Neurol. & Psychiat. **23**:715 (April) 1930.

2. Brickner, R. M.: Studies on the Pathogenesis of Multiple Sclerosis: II. Evidence of the Presence of an Abnormal Lipase in the Blood in Multiple Sclerosis, Bull. Neurol. Inst., New York **1**:105 (Jan.) 1931.

3. Crandall, L. A., and Cherry, I. S.: Presence of an Olive Oil Splitting Lipase in the Blood of Patients with Multiple Sclerosis, Proc. Soc. Exper. Biol. & Med. **28**:572, 1931.

using olive oil in place of lecithin. As it is known that many blood lipases are inactivated by quinine, it was decided to test the effects of this drug on patients.

Accurate studies coordinating the various clinical stages with different pathologic phases of the disease are lacking, and it is difficult to visualize in pathologic terms the clinical manifestations in patients with multiple sclerosis. The lack of this information becomes particularly troublesome when one is faced with an array of symptoms, some of which recede while the patient is under treatment, and some of which remain stationary. Therefore, in attempting to comprehend pathologically the behavior of the different symptoms, one is forced to rely on theoretical considerations. The theoretical background that has been found useful in analyzing the observations made in the present study is outlined in the subsequent paragraphs.

During the usual course of multiple sclerosis, it is assumed that myelin is acted on and disintegrated by some extraneous agent. While this agent is present and not successfully combated, myelin is being destroyed. At times when the agent is either absent, or is present but is being adequately repressed, myelin is, presumably, not being destroyed, and new myelin may be deposited. Thus, there may be a frequent shifting of the balance in the amount of myelin that is covering each axon.

When myelin disintegrates, its products, acting as foreign bodies, excite a phagocytic reaction that is followed by gliosis. Obviously, the longer the period of disintegration, the greater in amount will be the gliosis and the heavier will be the subsequent scar. However, not only the duration of the disintegrative process but also its rapidity should determine the heaviness of the scar, so that a very rapid myelinolysis of two months' standing might result in as dense a scar as that from a much slower disintegration of eighteen months' existence. These are important considerations, since the chances for improvement of a given lesion probably depend mainly on the density of the scar; the lighter the scar, the better, theoretically, should be the likelihood of myelin replacement.

The disintegration, myelin replacement and scarring should also be thought of as occurring simultaneously in various parts of the central nervous system, in greater and lesser stages of advancement. The chance for relatively complete myelin replacement in any given lesion must depend, in part, on the degree to which scarring has occurred, so that in any particular patient the possibility that healing will occur should vary for the different lesions.

There is another, less theoretical phase of the pathology of the disease which has a distinct bearing on the outcome with respect to each lesion. In late stages, many of the denuded axis cylinders, hitherto

viable, degenerate and their cells of origin die. Such lesions, of course, are no longer those of active multiple sclerosis, but are end-products that are not amenable to treatment.

It is therefore of first importance to separate the symptoms of supposedly heavily scarred lesions from those presumably due to early myelinolysis. This separation cannot be made by considering the patient as a whole; hence, it is a primary requirement to regard the symptoms in any given case individually rather than as an ensemble. In no other way can any form of therapy be adequately tested, and by no other means can one ultimately achieve reliable prognostic signs.

TECHNIC OF ADMINISTRATION OF QUININE

More complete details concerning the technic of administration are given in a previous communication.[4]

Dosage.—It has seemed wisest to use the hydrochloride of quinine because it is the most soluble of the readily available preparations. The dosage has been 5 grains (0.32 Gm.), three times a day, by mouth, unless sensitiveness to the drug has necessitated the employment of a smaller dose. Frequently, patients have been unable to tolerate 15 grains (0.97 Gm.) a day at the beginning; in such cases the medication has been omitted, to be given again four days later. Usually, 2½ grains (0.16 Gm.), two or three times a day, will be tolerated, and it is generally possible slowly to work back to three 5 grain doses. As with other forms of medication the proper dose must be separately determined for each patient. During the occurrence of cinchonism (manifested by ringing or a feeling of stuffiness in the ears, deafness, headache or nausea), many of the patients have shown a relapse of all symptoms, with recuperation on omission of the drug. It must be readministered to prevent a second regression.

After improvement has set in and has reached a more or less constant level, it has seemed wise to interrupt the administration of the drug frequently, even in the absence of cinchonism. Sometimes, under these conditions, symptoms will improve still further in the first three or four days without quinine, after which they begin to regress; at that time the medication should be reinstated. The length of these periods must be determined individually for each patient.

Duration of Administration.—According to the hypothesis, quinine should not cure the disease but should merely depress the activity of the myelinolytic agent. Therefore, the drug should, theoretically, be taken indefinitely. The observations have been in progress for not much more than a year, and it is not yet possible to say whether the quinine can be omitted for any great length of time.

Period of Trial.—While, as will be seen, most of the improvements start within from two to four weeks, some of the older symptoms have improved only after longer periods (from six to eight months), and it is suggested that in any case the drug should not be abandoned until after at least ten months of trial.

Regressions During Treatment.—Regressions during treatment need not be discouraging. An early regression may precede marked improvement; a later one may mean that the patient has had too much quinine. In either event, the quinine

4. Brickner, R. M.: The Administration of Quinine Hydrochloride in Multiple Sclerosis, New York State J. Med. **31**:885 (July 15) 1931.

should be omitted for from two to four days; usually there will be a prompt return to the old status, with increased improvement when the administration of the drug is resumed. Occasionally, a regression may indicate that the dosage should be increased; this is usually revealed by exaggerated decline when the medication is stopped. It has also been found that a transient regression usually occurs during the courses of infections, such as colds, boils, etc.

Special Considerations.—All patients on quinine therapy must be kept under continuous observation. They should be seen at a minimum of once a week. Every change, both favorable and unfavorable, should be reported at once; otherwise the constantly changing indications for interrupting and reinstating the medication and for changing its dosage will be overlooked. Careful notes should be kept, so that particular idiosyncrasies may be known. The details of treatment have rarely been the same for any two patients.

RESULTS

The present report is of sixteen well established cases of multiple sclerosis. One patient was treated by Dr. George Hyslop and one by Dr. Gerald Parker; of the remaining fourteen, who were treated by me, one was referred by Dr. James Huddleson and one by Dr. Louis Hausman. The results are tabulated according to symptoms.

In view of the difficulties in evaluating therapeutic results it might well be required of any medication that, for the demonstration of specificity, improvement in symptoms should appear within a certain given limit of time. If the relative fixity of such an interval can be established for a reasonable proportion of the symptoms, it is fair evidence that there is some specificity in the effect of the drug. Such is the case with quinine hydrochloride. The average interval between the commencement of treatment and the first evidences of improvement has been about four weeks.

Occasionally, one or more symptoms have improved within the first week of therapy; in other instances there has been no sign of change for five or six weeks or longer. As would be anticipated, the symptoms do not all begin to recede at the same time; it is usual for those of shortest duration to be the first to change. An important consideration in this connection is the fact that the first improvements may be of symptoms such as tingling in one hand, which are of minor consequence to the patient. The persistence of a disability as great as blindness or spasticity of the lower extremities after other improvements have begun to occur should, of course, not be discouraging.

The symptoms included various forms of disturbance of gait due to spasticity, paresis, paralysis or ataxia, intention tremor, frequency or hesitancy of urination, impotence, hypesthesias and dysesthesias, aphasia, scanning speech, blindness, diplopia, facial paralysis, abducens paralysis, fatigability and forced laughter. It has been found that the character of the symptom has nothing to do with the outcome. As has

been stated, the likelihood of improvement of a symptom depends, in large part, on its age.

In the accompanying table the duration of the symptoms is listed, together with the incidence or absence of improvement.

It should be stressed that of twenty-five symptoms of twelve months' duration or less, twenty-one improved; of thirty-two with durations not greater than twenty-four months, twenty-eight improved. In addition, it was observed that the shorter the duration of the symptom the more striking was its improvement.

Many symptoms, such as intention tremor and disturbed gait, showed marked improvement. Objective improvements of this type were many. In five cases improvement also occurred in the objective sensory symptoms; in one case a complete paralysis of the left abducens

Duration of Symptoms, with Incidence or Absence of Improvement

Duration	Number of Symptoms That Have	
	Improved	Not Improved
Six months or less	6	1
Six to twelve months	15	3
Twelve to eighteen months	4	0
Eighteen to twenty-four months	3	0
Not long; exact duration uncertain	4	0
Two to three years	5	5
Three to four years	2	4
Four to five years	0	6
Five to six years	1	4
Nine to ten years	0	1
Ten to thirteen years	0	3
Many years; exact duration uncertain	0	6
Total	40	33

and facial nerves disappeared; in one, nystagmus was no longer observable; in one, the lower abdominal reflexes reappeared, and in one there was disappearance of a permanent right ankle clonus. It has not been possible to learn the duration of most of these signs.

In three cases there was slight regression of some of the symptoms at the beginning of treatment. These symptoms were among those that later made marked improvement. Thus, an initial slump may be a favorable sign of the activity of the drug. New, transitory dysesthesias also occurred several times during the period of active improvement. I have no good explanation to offer for these phenomena; possibly they may be interpreted as representing disturbed function of neurons due to the new laying down of periaxonal myelin.

All of the improvements except three have been maintained at a fairly constant level up to the present writing. In these three instances there was permanent regression of individual symptoms while treatment was going on, which had not previously improved. These must stand

as essentially unexplained. Otherwise, even among the symptoms that failed to improve, no regressions were noted.

The various patients have been receiving treatment with quinine for periods of a month to a year.

The present evaluation of the effects of quinine in the course of multiple sclerosis must be made from two standpoints, that of the patient, who is interested in cure, and that of the investigator, who is also interested in cure, but, as well, in the theoretical aspects. The evidence appears to be good that there is something specific in the action of quinine hydrochloride; there seems to be no other interpretation of the relative fixity of the period of treatment prior to improvement, nor of the definite grouping, by duration, of symptoms which have and have not improved and which have improved to greater and lesser degrees. This specificity, if borne out by additional work, is of theoretical importance, since it indicates that the myelin is acted on not only by an agent external to it but by one the activity of which can be depressed by quinine. From the patient's point of view the results so far may also be said to be favorable, but since the maximum period of observation has been only a year nothing can be said, at the moment, of the value of quinine to the patient over a very long interval. Not only will more cases have to be studied, but there will, of course, have to be a much longer period of observation.

As they stand, the results seem to warrant additional study. It would be advisable, at present, that any such investigations should be undertaken only in unquestioned cases of multiple sclerosis and that the study be made symptom by symptom, with detailed notes.

Certain of the patients had made previous improvements, some of which had been spontaneous and some of which had occurred during various forms of treatment. Most of these were well in the past and therefore had no direct bearing, in themselves, on the results of the present therapy.

SUMMARY

1. Experiences with the use of quinine hydrochloride in the treatment of sixteen patients with multiple sclerosis over a period of one year are given.

2. The theory of quinine therapy rests on the hypothesis, suggested but not yet proved, that the lesions are caused by abnormal lipolytic activity of the blood, and that this activity may be due to the presence of an abnormal lipase.

3. The effects of the therapy have been studied symptom by symptom.

4. Forty symptoms, most of them of short duration, have shown marked improvement. Thirty-three symptoms have not improved; most of the latter were old.

5. All of the improvements but one have been maintained at a fairly constant level up to the present writing. Only three of the unimproved symptoms have regressed.

6. The final estimation of the actual therapeutic value of quinine in this disease cannot be made until more cases have been studied over a longer period. However, specificity in the effect of quinine is suggested by: (*a*) the relative fixity of the interval between the initiation of treatment and the beginning of improvement; (*b*) the grouping, by duration, of symptoms which have and have not improved; (*c*) the fact that the most recent symptoms have improved not only the most promptly, but also the most completely.

MULTIPLE TUMORS OF THE NERVOUS SYSTEM

REPORT OF A CASE

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Multiple tumors of the nervous system in which each separate tumor has somewhat different histologic and functional characteristics occur with sufficient frequency to warrant a more thorough study of them. An etiologic classification of such tumors would remove the obvious defects in our present method of attempting to classify them according to their similarity to adult tissues (Masson [1]). The following case is a striking example.

REPORT OF CASE

Clinical History.—A. M., a white man, aged 30, a janitor and trucker, was admitted to the Jefferson Hospital on Feb. 15, 1930, to the service of Dr. Edward Strecker. The father died of insanity at 65, and one brother committed suicide. The patient had had a congenital strabismus from birth. The present illness came on gradually. First there was ataxia and difficulty in raising the feet to walk up steps, with gradually increasing intermittent edema and pain across the abdomen. A few days after admission, a fleeting ankle clonus developed, and the reflexes of the lower extremities became diminished. Examination of the eyegrounds showed that the disks were blurred; the veins were full, the arteries small and the whole retina swollen; the swelling of each disk was 2 diopters. The visual fields were slightly contracted; the blind spot was enlarged to three times its normal size. The color fields were greatly contracted. These changes became more accentuated. Vision decreased to 20/200 in each eye. A Bárány test suggested a lesion of the mesial side of the right cerebellum. During May, the deep and abdominal reflexes became absent. Both legs and hands were numb; urinary incontinence developed. When a spinal tap was made at this time, the fluid was under 12 mm. of pressure and was xanthochromatic. Headaches became more intense. Later, the right leg became much weaker than the left, with definite ankle clonus. Adiadokokinesis of both hands was present. The papilledema lessened. The vision improved to 20/80 on the right and 20/50 on the left, and the visual fields enlarged for colors. Wassermann and Kahn tests of the spinal fluid and blood were negative. There was a persistent trace of albumin in all specimens of urine. A complete blood count and chemical examination of the blood gave normal results. The systolic blood pressure ranged between 120 and 140; the pulse rate was constantly at 120;

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A grant from the Martin Research Fund aided in this work.

1. Masson, P.: Diagnostics de laboratoire: II. Tumeurs, Paris, Norbert Maloine, 1923.

the respirations were from 26 to 30; the temperature was between 97 and 101 F., with several records above normal. On October 22, signs and symptoms of pneumonia developed, and the patient died five days later.

Postmortem Examination.—Six tumors were found. One, designated as the sciatic nerve tumor, was attached to the sheath covering the right lumbosacral trunk and sciatic nerve. It was oblong, retroperitoneal and measured 10 by 7 by 3 cm. It extended from the intervertebral foramen beneath the fourth lumbar vertebra to the great sciatic notch. The foramen was dilated, and the bone bounding it was eroded and rough. The tumor was encapsulated and on section consisted of pinkish-white lobulated tissue in which there were large areas of necrosis and hemorrhage.

Fig. 1.—Photomicrograph showing section of tumor of the sciatic nerve; × 100.

A second tumor, designated as the arachnoid tumor, occupied a position over the upper portion of the right motor area of the cerebrum between the dura and arachnoid, to each of which it was firmly attached. It was flattened, measured 4 by 2.5 by 1.5 cm., was well defined and encapsulated and on section firm and pinkish white. The overlying bone was normal. The brain was not infiltrated.

A third, designated as the tumor of the pons, lay entirely within the pons and medulla. It measured 4.5 by 1 by 0.5 cm., was sharply circumscribed, white and slightly lobulated. The brain stem was distorted and atrophied but not infiltrated.

A fourth, the pituitary tumor, was attached to the left of the pituitary body, the left posterior communicating branch of the circle of Willis and a nerve. It was flattened, measuring 1 by 1 by 0.2 cm., firm, white and sharply circumscribed.

A fifth, attached to the sheath of the intracranial portion of the motor oculi nerve, was spherical, measuring 1 cm. in diameter, firm, encapsulated and on section lobulated and white.

The sixth, similar in size and appearance to the fifth tumor, was attached to the third right cervical nerve within the canal.

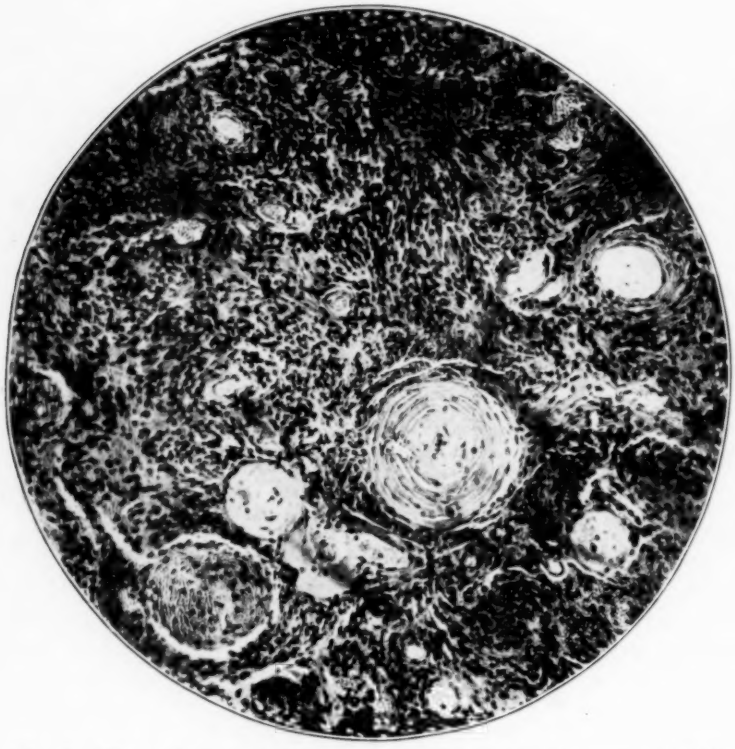

Fig. 2.—Photomicrograph showing section of arachnoid tumor; × 100.

Additional features were dilatation of the ventricles of the brain, hydroureter on the right and suppurative bronchopneumonia.

Method of Study of the Tumors.—Thin sections from the first, second, third and fourth tumors were fixed in a diluted solutions of formaldehyde, U. S. P. (1 : 10) blocked in paraffin, cut and stained with hematoxylin and eosin, eosin-methylene blue, van Gieson's stain for collagen, Verhoeff's stain for elastic tissue, Perdrau's silver impregnation reticulum stain and phosphotungstic acid-hematoxylin stain for fibroglia fibers.

Description of Tumors.—The sciatic nerve tumor (fig. 1.) consisted of closely packed uniform, spindle-shaped cells, with deeply stained nuclei, grouped together in small bundles which ran in every direction. The vessels were well formed but not numerous. Large areas of necrosis and hemorrhage were present. Elastic,

collagen and silver-staining fibers were present in great abundance everywhere between the cells. There were few fibroglia fibers.

The arachnoid tumor (fig. 2.) was very vascular and cellular. Mitotic figures were found with ease. The cells were spindle-shaped and slightly oval; their nuclei were vesicular and rich in chromatin material. Their cytoplasm was abundant and finely granular. Numerous concentrically arranged whorls were present. Some of them were composed entirely of cells; others of cells and hyaline material in varying proportions. The more cellular ones had a blood vessel in the

Fig. 3.—Photomicrograph showing section of tumor of pons; × 100.

center. Small areas of necrosis were infrequently found. The hyalinized whorls consisted of collagen, elastic, fibroglia and silver-staining fibers. About the cells were a few fibroglia and collagen fibers.

The tumor of the pons (fig. 3) consisted of spindle cells arranged singly and in sheets, widely separated by a clear hyaline substance. The cell outlines were frequently indistinct. The nuclei were deeply stained. Mitosis was rare. Great numbers of fibroglia fibers were present everywhere. Collagen, elastic tissue and silver-staining fibers were absent, except for a few about blood vessels.

The pituitary tumor (fig. 4) consisted of lobules of tumor cells, separated from each other by trabeculae formed of vascular adult connective tissue containing collagen fibroglia, elastic and silver-staining fibers. The tumor cells of the lobules were characteristically arranged in syncytial masses, immediately surrounded by

a clear area. When single cells could be studied, they were round or oval. Their nuclei were pale and vesicular, with little chromatin except around the nuclear membrane. Cytoplasm was abundant and finely granular. No fibers could be demonstrated about these cells. The vessels in the trabeculae were thin-walled, irregularly widened and narrowed channels, lined by a single layer of flattened endothelium, which in places shaded off into tumor cells. Many of the vessels were empty. Others contained coagulated serum, red blood cells and leukocytes. In a few, masses of tumor cells were present.

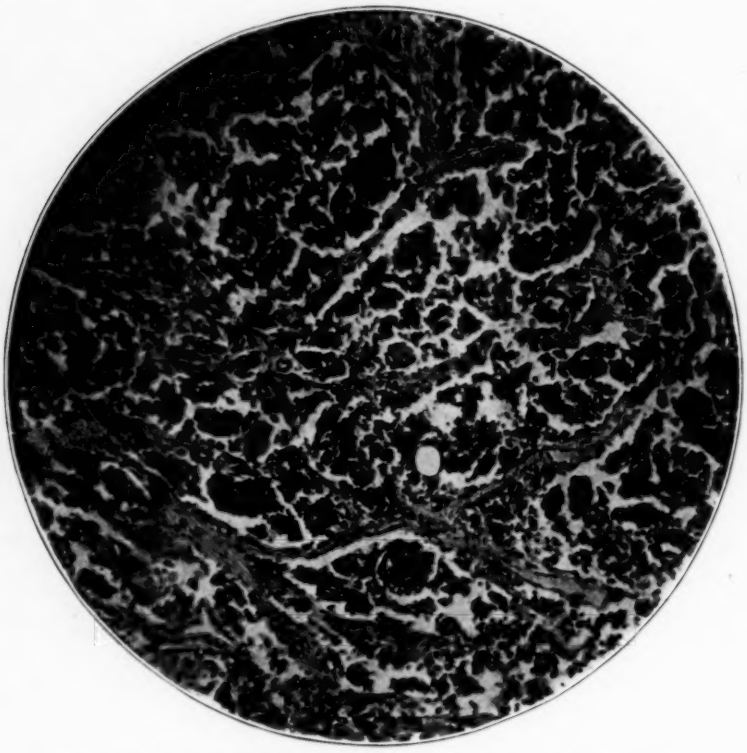

Fig. 4.—Photomicrograph showing section of pituitary tumor.

COMMENT

Systematic comparison of these tumors each with the other shows striking differences.

The sciatic nerve tumor differed from the arachnoid tumor by having more regularly spindle-shaped and uniform cells arranged in bundles and not dividing, an absence of whorls, many more elastic, collagen and silver-staining fibers; fewer blood vessels, and larger areas of necrosis and hemorrhage. It differed from the tumor of the pons by being more cellular, less vascular and more necrotic; having an abundance of elastic, collagen and silver-staining fibers; in the

arrangement of the cells into bands, which ran in every direction, and by an absence of hyaline material between the cells. It differed from the pituitary tumor in size, shape, staining, and number and arrangement of cells; in the method of lobulation; the number, construction and contents of the blood vessels, and by the presence of elastic, collagen, fibroglia and silver-staining fibers between the cells, which were entirely absent in the latter.

The arachnoid tumor differed from the tumor of the pons by having whorl formation; a different arrangement and less regularity in the size, shape and staining of its cells; many more mitotic figures; a greater number of differently arranged blood vessels; no hyaline areas separating the cells; small areas of necrosis; the presence of collagen, but fewer fibroglia fibers. It differed from the pituitary tumor by having whorl formation; in the type and arrangement of its cells; in the construction, number and arrangement of blood vessels, and in having fibroglia and collagen fibers between the cells.

The tumor of the pons differed from the pituitary tumor in type and arrangement of cells; in the separation of its cells by hyaline areas; in the construction, number and arrangement of blood vessels, and by the presence of great numbers of fibroglia fibers between the cells and in lobulation.

The arachnoid tumor can be classified as a dural endothelioma, meningioma (Cushing [2]) or meningothelioma, psammomatous type (Bailey and Bucy [3]). All these terms are synonymous.

Schmidt [4] pointed out the relationship between the pacchionian bodies and the origin of these tumors. Winkelman and Wilson [5] have demonstrated the possible relationship of such tumors to localized proliferations of the arachnoid. Flexner [6] has shown that the pacchionian bodies originate from the arachnoid, which previously split off from the dura as one layer of fibroblastic cells. Utilizing these facts and his own methods, Mallory [7] demonstrated that embryologically, morphologically

2. Cushing, H.: The Meningiomas (Dural Endotheliomas), Brain **45**:282, 1922.

3. Bailey, P., and Bucy, P. C.: Origin and Nature of Meningeal Tumors, Am. J. Cancer **15**:15 (Jan.) 1931.

4. Schmidt, M. B.: Ueber die Pacchionischen Granulationen und ihr Verhältniss zu den Sarcomen und Psammomen der Dura mater, Virchows Arch. f. path. Anat. **170**:429, 1902.

5. Winkelman, N. W., and Wilson, George: Localized Proliferations of the Arachnoid, Possible Relation to the Origin of Tumors, Arch. Neurol. & Psychiat. **14**:46 (July) 1925.

6. Flexner, L. B.: The Development of the Meninges in Amphibia: A Study of Normal and Experimental Animals, publ. 394, Contrib. Embryol. no. 22, Carnegie Inst., Washington, 1929, p. 31.

7. Mallory, F. B.: The Type of Cell of the So-called Dural Endothelioma, J. M. Research **41**:349, 1920.

and functionally, these tumors are fibroblastic in nature. In favor of this view are Van Wagenen, [8] Coupin, [9] Snessarew [10] and Hasoi.[11] Opposed to it are Oberling,[12] Lhermitte,[13] Roussy and Cornil [14] and Harvey and Burr,[15] who believe the leptomeninx to be neuro-ectodermal in origin and the tumors arising from it neuro-epithelial in type.

The tumor attached to the sheath of the sciatic nerve can be classified as a perineural fibroma. Mallory believes the perineurium to be a direct outgrowth of the arachnoid or an analogous differentiation of the mesenchyme around the nerves. Hassin [16] has demonstrated the analogy between the cerebrospinal and spinal arachnoid villi, so that arising from either source the tumor has the same embryologic origin as the meningioma. However, its adult structure is considerably different. It is more distinctly fibroblastic.

The tumor of the pons, which was completely surrounded by the brain stem, had cells similar to adult fibroblasts which were surrounded by fibroglia fibers. These differed from adult fibroblasts in that they manufactured no collagen, elastic or silver-staining fibers. To account for a fibroblastic origin there would have to have been an inclusion of the leptomeninx in the brain stem.

The tumor of the pituitary has some characteristics similar to the meningothelioma of Bailey and Bucy, and others similar to the angioblastic type (tumors described by Bailey and Cushing formed of cells like the angioblast of Sabin [17]). But the differences are too great. From

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8. Van Wagenen, W. P.: Elastic Tissue in Meningeal Fibroblastomas, So-called "Dural Endotheliomas," Arch. Surg. **18**:1621 (April) 1929.

9. Coupin, F.: Les formations choroidiennes des poissons, Arch. de morphol. gén. et expér. **20**:1, 1924.

10. Snessarew, P.: Ueber die Pigmentzellen der Piae matris beim Menschen, ihren Zusammenhang mit den Nervenfasern, ihr Genese und Funktion, Ztschr. f. Zellforsch. u. mikr. Anat. **9**:683, 1929.

11. Hasoi, Kyoshi: Meningioma, with Special Reference to the Multiple Intracranial Type, Am. J. Path. **6**:245, 1930.

12. Oberling, C.: Les tumeurs des méninges, Bull. Assoc. franç. p. l'étude du cancer **11**:365, 1922. La Gliomatose méningo-encéphalique, Bull. et mém. Soc. anat. de Paris **94**:334, 1924.

13. Lhermitte, J., and Leroux: Gliomes typiques et atypiques des nerfs périphériques, Bull. Assoc. franç. p. l'étude du cancer **9**:112, 1920.

14. Roussy, G., and Cornil, L.: Les tumeurs méningées; revue critique, Ann. d'anat. path. **2**:63, 1925; A propos de la conception et de la classification des tumeurs cérébrales, Rev. neurol. **2**:4, 1928.

15. Harvey, Samuel C., and Burr, Harold S.: An Experimental Study of the Origin of the Meninges, Proc. Soc. Exper. Biol. & Med. **22**:52, 1924.

16. Hassin, George B.: Villi (Pacchionian Bodies) of the Spinal Arachnoid, Arch. Neurol. & Psychiat. **23**:65 (Jan.) 1930.

17. Sabin, F. R.: Preliminary Note on the Differentiation of Angioblasts and the Method by which They Produce Blood Vessels, Blood Plasma and Red Blood Cells, as Seen in the Living Chick, Anat. Rec. **13**:199, 1917.

its evident origin either from the pacchionian bodies about the hypophysis, where they are numerous (Aoygi and Kyuno[18]), or from the leptomeninx it might be considered fibroblastic in nature. If this is the case, metaplastic changes must have occurred to account for such striking clearcut distinctions between its cells and normal fibroblasts.

Because of the differences in these tumors no one can be considered as the original tumor which by metastasis gave rise to the others. Bailey and Bucy showed that there were differences in a tumor examined on two different occasions after an interval of months. But Penfield[19] did not believe that this occurs with any degree of frequency. To account for the tumors in this case there must have been one etiologic agent acting on a congenitally impaired embryologic tissue. And as these tumors arose from structures developing from either the neuroepithelium or the mesenchyme, the fault must have been in one or the other (Penfield[19] and Castellino[20]).

There is the suggestion here that this case is a rare form of von Recklinghausen's disease[21] in which the common manifestation of multiple superficial fibromas was absent. It has been pointed out that the disease may be complicated by tumors of peripheral and optic nerves, spinal cord (Parker[22]), meningiomas (Mallory[7]), tumors of the endocrine gland (Castellino[20]) and by ependymomas and astrocytomas (Penfield[19]). But until clearcut intermediary forms are shown to occur, the tumors in the case reported cannot be diagnosed thus.

CONCLUSION

A case of multiple tumors of the nervous system is reported in which each differed from the other in the type and arrangement of the cells, number of mitotic figures, construction and number of blood vessels, amount of necrosis and kind and quantity of fibers manufactured. Evidence is presented that all these tumors originated from the same embryologic tissue.

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18. Aoygi and Kyuno, in Cushing (footnote 2).

19. Penfield, W., and Young, A. W.: The Nature of von Recklinghausen's Disease and the Tumors Associated with It, Arch. Neurol. & Psychiat. **23**:320 (Feb.) 1930. Penfield, W.: Cranial and Intracranial Endotheliomata: Hemicraniosis, Surg., Gynec. & Obst. **36**:657, 1923; The Encapsulated Tumors of the Nervous System, ibid. **45**:178, 1927.

20. Castellino, P. G.: Familial Recklinghausen's Disease: A Clinical and Pathogenetic Contribution, Urol. & Cutan. Rev. **34**:384, 1930.

21. von Recklinghausen: Ueber die multiplen Fibromen der Haut und ihre Beziehung an den multiplen Neuromen, Berlin, A. Hirschwald, 1882.

22. Parker, H.: A Case of Reckinghausen's Disease with Involvement of the Peripheral Nerves, Optic Nerve and Spinal Cord, J. Nerv. & Ment. Dis. **46**:441, 1922.

CEREBRAL CIRCULATION

AFFERENT IMPULSES FROM THE BLOOD VESSELS OF THE PIA

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AND
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Since the cerebral circulation is dominated by the blood vessels of the pia mater, their activity is of major importance to the brain. That these vessels are richly supplied with nerves can no longer be questioned. Convincing data are available from both the histologic and the physiologic points of view.

Histologically, it has been demonstrated that these vessels have about them and in their walls many nerve fibers and end-organs. Stöhr [1] differentiated them into motor and sensory components, but Hassin [2] did not feel justified in making this distinction. They agreed in stressing the significance of this nerve supply because of the importance of the pial vessels. Penfield [3] found that not only the pial vessels, but also their intracerebral branches, are equipped with nerves.

Physiologically, the knowledge of the function of these nerves and end-organs has been more or less limited to the demonstration of motor effects, viz., that the pial vessels are capable of responding to motor nerve impulses by constriction and dilatation. Specifically, constriction may occur through stimulation of the cervical sympathetic, and dilatation through stimulation of the vagus-depressor nerve.[4] As to the possibility of a sensory function of some of these nerves, little conclusive evidence is available. Therefore, to round out the story, we have attempted to

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Communication read at the International Neurological Congress, Bern, Switzerland, Sept. 4, 1931.

From the Psychobiological Laboratory, Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital.

1. Stöhr, P.: Mikroskopische Anatomie des vegetativen Nervensystems, Berlin, Julius Springer, 1928, p. 177.

2. Hassin, G. B.: The Nerve Supply of the Cerebral Blood Vessels, in The Vegetative Nervous System, Baltimore, Williams & Wilkins, 1930, p. 437.

3. Penfield, W.: Intracerebral Vascular Nerves, Arch. Neurol. & Psychiat. **27**:30 (Jan.) 1932.

4. Forbes, H. S., and Wolff, H. G.: The Cerebral Circulation: III. The Vasomotor Control of the Cerebral Vessels, Arch. Neurol & Psychiat. **19**:1057 (June) 1928.

demonstrate physiologically that this nerve supply has sensory as well as motor functions.

To do this, it was necessary, first, to collect evidence of a functional nature of the presence of afferent nerves in the pia, and second, to determine the distribution of these afferent nerves in terms of their relation to blood vessels and to those portions of the pia which apparently are free of blood vessels. We therefore attempted to explore the pia over the surface of the cerebral cortex with minimal faradic currents, in animals operated on under local anesthesia.

A demonstration of the presence of sensory end-organs or fibers over the cerebral cortex has been difficult because of the inadequacy of the methods of detection of response to sensory stimulation. In man, attempts have been made to detect response to faradic stimulation [5] and to operative manipulation [6] of the blood vessels and pia, by having patients during an operation report on their awareness of the stimulation. Contradictory results have been obtained, but the negative results obtained in this way are not satisfactory evidence of the absence of responses, for two reasons: (1) One cannot rely on the report of awareness of sensory stimulation as the sole criterion of a response, since it is conceivable that minimal faradic stimulation may produce responses that do not involve awareness of the stimulation; (2) operative manipulation may not provide adequate stimuli for these sensory structures. In man and in experimental animals, attempts have been made to detect response to stimulation by observation of such phenomena as crying, withdrawal from the stimulus, and respiratory,[7] blood pressure and pulse changes, but again these are not delicate enough for the study of the effects of minimal stimulation. In the experiments here presented, this difficulty was in large part overcome by making use of the galvanic skin reaction to demonstrate response to stimulation. This has proved to be a reasonably sensitive method.

The galvanic skin response may be evoked in the intact animal by a sudden noise or a painful stimulus, e. g., sounding a horn, or pinching the tail. In man, it may be evoked by a pin-prick or by a word that arouses an emotional response, as in the "psychogalvanic association tests" used in psychiatric practice.[8] It may be present when there are

5. Penfield, W.: Personal communication.

6. Stöhr (footnote 1). Hassin (footnote 2).

7. Hitz, J. B., and Kammer, A. G.: The Effects of Stimulation of Cerebral Blood Vessels, quoted by Leake, J. P.; Loevenhart, A. S., and Muehlberger, C. W.: Dilatation of Cerebral Blood Vessels as a Factor in Headache, J. A. M. A. **88**:1076 (April 2) 1927.

8. References to the literature on galvanic skin responses are collected in Landis, C., and DeWick, H. N.: The Electrical Phenomena of the Skin, Psychol. Bull. **26**:64, 1929.

no other observable data from which one can conclude that the stimulation is producing an effect. For example, the application of a painful stimulus to an area of skin that has been rendered anesthetic by suggestion in hypnosis results in a galvanic skin response even though the patient shows no facial or bodily expression of pain and is unable to report any awareness of the stimulation.[9] The usefulness of the galvanic skin response is enhanced by the fact that the experimenter is not dependent on the cooperation of the subject as witness. Hence its value to indicate responses obtained by stimulation of afferent pathways and end-organs in experimental animals.

One serious limitation to the use of this method is that the galvanic skin response is considerably reduced in general anesthesia.[10] And yet, for finer exploration over the cerebral cortex, immobilization is a prerequisite. Bulbocapnine proved to be a fortunate drug for our purpose, as it produces almost complete immobilization and apparently does not interfere with sensory reception, with nerve conduction or with the galvanic response. It may actually augment the galvanic response.[10] In combination with procaine hydrochloride as local anesthetic, the operations could be performed quickly and with minimal discomfort and shock to the animals.

METHODS

Young adult cats, weighing from 2.5 to 3.5 Kg., were used as experimental animals. Two operative methods were utilized. The first method (six animals) involved a two-stage operation: (*a*) Craniotomy was performed under ether, exposing the right occipital, parietal and temporal regions; (*b*) forty-eight hours later, under bulbocapnine and procaine hydrochloride, the incision was opened and the dura turned back. From 50 to 60 mg. of bulbocapnine (Merck), given subcutaneously, was found to be an effective dose for operative purposes. Procaine hydrochloride was used as indicated as a local anesthetic, care being taken to prevent its coming into contact with the dura or pia. The second method (ten animals) involved but a single operation, since it was found that the entire operation could be performed under bulbocapnine and procaine hydrochloride without causing the animal any pain, and apparently without reducing the sensitivity of the animal to minimal electrical stimulation.

A unipolar stimulating electrode was used for exploration. It consisted of a platinum needle with a spherical tip (about 0.5 mm. in diameter) in circuit with a DuBois-Réymond induction coil and two dry cell batteries. The neutral electrode, placed on the shaved abdomen, consisted of a zinc plate covered by a paste of kaolin mixed with saturated zinc sulphate solution.

After the pia mater was exposed, the unipolar electrode was placed on various parts of the pial surface, and faradic stimulation, of a duration of a second or less, was applied. The motor area was avoided because of the complications resulting from motor disturbances when this area is stimulated. The areas adjacent to the motor cortex were avoided because stimulation of certain of these

9. Levine, M.: Psychogalvanic Reaction to Painful Stimuli in Hypnotic and Hysterical Anesthesia, Bull. Johns Hopkins Hosp. **46**:331 (May) 1930.

10. Richter, C. P.: Personal communication.

cortical areas gives galvanic skin responses.[11] These are probably the only areas where stimulation produces such reactions.[11] The avoidance of these areas minimizes the likelihood that, in the present experiments, the galvanic skin responses were due to stimulation of the cortex itself. Therefore the responses obtained probably are in most cases responses to stimulation of blood vessels and pia.

No attempt was made to determine if the stimulation was affecting specific sense organs or was affecting nerve fibers. It is likely that both were involved in the stimulation.

The strength of the current to be used was determined by moving the secondary coil gradually toward the primary coil until stimulation of a blood vessel elicited a galvanic skin response that was not accompanied by skeletal muscle movements. The pial surface was then explored, each stimulus being automatically recorded on the photographic record of the galvanic responses.

Records of the galvanic skin response were obtained by photographing the deflections of the string of a galvanometer attached to the experimental animal (apparatus described by Richter[12]). The wires leading to the galvanometer were attached to relatively nonpolarizable electrodes (zinc disks covered with kaolin mixed with saturated zinc sulphate solution). The electrodes were applied to the skin of the large central pad of the cat's paw and to the pinna of one ear. The skin of the ear was previously pierced with a needle to eliminate responses from the ear, so as to obtain responses from the paw only. No external current was introduced after stimulation was begun, so that the currents given off had their origin in the activity of the skin of the pad of the paw (presumably of the sweat glands).[11]

Before each experiment the string of the galvanometer was so standardized (without the animal in the circuit) that the introduction into the circuit of 1 millivolt corresponded to a 10 mm. deflection of the shadow of the galvanometer string on the scale of the camera. Then, with the animal in the circuit, the skin resistance was measured by recording the number of millivolts necessary to deflect the string 10 mm. After the skin resistance was measured, the galvanometer string was so restandardized that the introduction of 10 millivolts caused a deflection of 25 mm. Subsequent to this standardization, no external current was used. Incidentally, it was observed that when the skin resistance was high, the animal would not show much galvanic skin responsiveness, perhaps because of an unfavorable reaction to operative manipulation. Under such circumstances, a short recuperative period was occasionally effectual in reducing the skin resistance, and with that the animal showed spontaneous galvanic skin fluctuations and gave a galvanic skin response to pinching the tail.

OBSERVATIONS

Faradic stimulation by the method just described produced galvanic skin responses. They were obtained from some points in all of the exposed areas. Galvanic responses were obtained with equal frequency from the various portions of the occipital, parietal and temporal areas explored. The frequency and magnitude of the responses depended on

11. Langworthy, O. R., and Richter, C. P.: The Influence of Efferent Cerebral Pathways upon the Sympathetic Nervous System, Brain **53**:178, 1930.

12. Richter, C. P.: A Study of the Electrical Skin Resistance and the Psychogalvanic Reflex in a Case of Unilateral Sweating, Brain **50**:216, 1927.

the number and size of the blood vessels in the area explored. The responses were most frequent and of greatest magnitude when the electrodes were applied to moderately large vessels (approximately 500 microns). Stimulation of arteries and veins gave similar results. The most common site at which stimulation gave deflections of greater magnitude was found to be at the junctions of large vessels.

Stimulation of certain portions of some vessels gave galvanic skin responses repeatedly, while stimulation of other portions of the same vessels gave no responses. However, it was found occasionally that some points on the vessels gave rise to a response on the first stimulation, but thereafter gave rise to no responses even on repeated stimulation. Further, after repeated stimulation of a responsive point on a blood

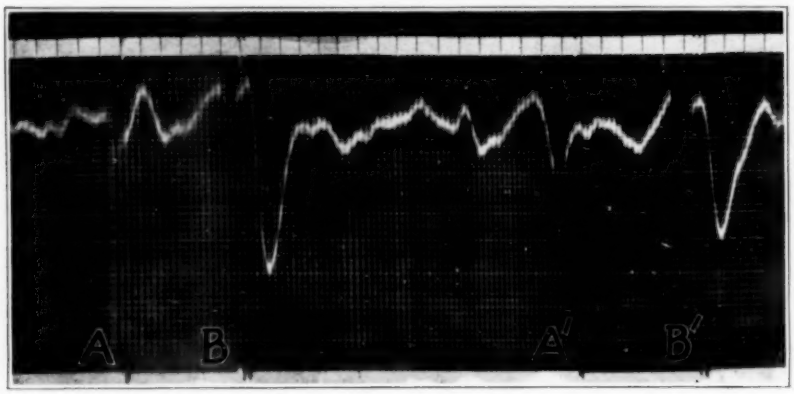

Fig. 1.—Photographic record of galvanic skin responses to minimal faradic stimulation of the pial vessels and of "intermediate pia." At *B* and *B'*, the blood vessels were stimulated, with large responses. At *A* and *A'*, the "intermediate pia" was stimulated, without response. Stimuli are indicated by signal marks at the bottom of the record. The time of stimulation of the "intermediate pia" is indicated by one signal mark. The time of stimulation of the blood vessels is indicated by the first of a pair of signal marks, i.e., the electrode was applied to the blood vessels at the time of the first only of a pair of signal marks, the second mark of the pair being merely for identification. Time is recorded in seconds at the top of the record.

vessel, the responses to stimulation of this point diminished or disappeared, to return only, if at all, after a considerable period of time.

One striking fact was that occasionally stimulation of the blood vessels resulted not only in a single galvanic skin response but also in moderately large repeated fluctuations indicating a longer and perhaps more marked response to stimulation. This occurred even when the first response was not large. This phenomenon was not observed after the stimulation of relatively nonvascular pia.

Occasionally, a marked galvanic skin response was accompanied or followed by irregular breathing and some movements of the skeletal muscles. Galvanic responses that followed such movements were not considered as direct responses to stimulation.

Stimulation of the relatively nonvascular pia between the vessels not infrequently gave small responses, particularly in those portions of the pia at a short distance from a vessel of moderate size. Here, too, as with the blood vessels, there were points that gave rise to responses on the first stimulation and therafter gave rise to no responses. It was possible that in this relatively nonvascular pia there were areas which were completely devoid of blood vessels and yet gave rise to galvanic responses. However, in these investigations no sharp differentiation was drawn

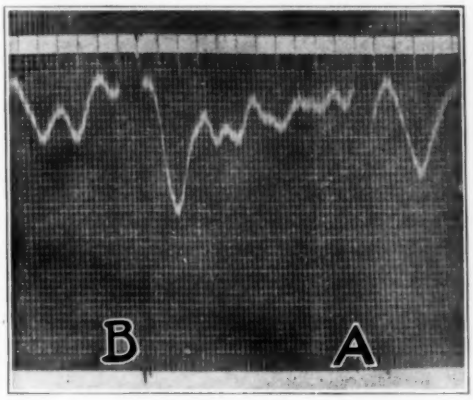

Fig. 2.—Photographic record of galvanic skin responses to minimal faradic stimulation, demonstrating moderate response to stimulation on the blood vessels at *B*, and smaller response to stimulation on the "intermediate pia" at *A*.

between completely nonvascular pia and that containing minute vessels. For convenience we have used the phrase "intermediate pia" in referring both to nonvascular pia and to pia containing minute blood vessels. We have used the phrase "pial vessels" in referring to the larger vessels, viz., arteries, arterioles, veins and venules, which are covered, of course, by pia.

All of the cats examined in this series gave galvanic skin responses, but not to an equal degree. In those that gave only a few galvanic responses, those few were always the result of stimulation of points on the blood vessels.

The pial blood vessels and the neighboring pia were stimulated 823 times. The blood vessels were stimulated 444 times and the "intermediate pia" 379 times. Of the 444 times when the blood vessels were stimulated, a galvanic skin response was obtained 175 times (38.9 per

cent). Of the 379 times when the "intermediate pia" was stimulated, a galvanic skin response was obtained 68 times (17.9 per cent). The responses were further classified as small, moderate and large. Of the 173 responses to stimulation of blood vessels, 81 were small (18.2 per cent of the 444 stimuli given); 75 were moderate (16.9 per cent of the 444 stimuli given), and 17 were large (3.8 per cent of the 444 stimuli given). Of the 68 responses to stimulation of "intermediate pia," 54 were small (14.3 per cent of the 379 stimuli given); 14 were moderate (3.6 per cent of the 379 stimuli given), and there was no large response (0 per cent of the 379 stimuli given).

To compare the responses obtained from blood vessel stimulation and "intermediate pia" stimulation, 38.9 per cent of the stimuli applied to blood vessels, evoked responses of various size, and only 17.9 per cent of the stimuli applied to "intermediate pia" evoked responses of various size. Further, 16.9 per cent of the stimuli applied to blood vessels

Summary of the Results of Galvanic Skin Responses to Minimal Faradic Stimulation of (1) Pial Blood Vessels, and (2) "Intermediate Pia" (Non-vascular Pia and Pia Containing Minute Vessels) *

	Pial Blood Vessels		"Intermediate Pia"	
	Number	Percentage	Number	Percentage
Total number of stimuli given.....	444	....	379	....
Galvanic skin responses of various size..	173	38.9	68	17.9
Galvanic skin responses of small size.....	81	18.2	54	14.3
Galvanic skin responses of moderate size	75	16.9	14	3.6
Galvanic skin responses of large size.....	17	3.8	0	0.0

* Total number of cats, 16; total number of stimuli, 823.

evoked responses of moderate size, while only 3.6 per cent of the stimuli applied to "intermediate pia" evoked responses of moderate size. Three and eight tenths per cent of the stimuli applied to blood vessels evoked large responses, and, in contrast, none of the stimuli applied to "intermediate pia" evoked large responses.

In general, it may be stated that stimulation of the pial blood vessels produced galvanic skin responses more than twice as often as did stimulation of the "intermediate pia"; further, stimulation of the pial blood vessels produced noticeably larger galvanic skin responses than did stimulation of the "intermediate pia."

In four cats, the exposed pial surface was painted with 1 or 2 per cent procaine hydrochloride after it had been demonstrated that galvanic responses of considerable magnitude were being elicited from certain points; after five or ten minutes, the responses to faradic stimulation were greatly reduced or entirely eliminated, although the galvanic skin response could still be elicited by pinching the tail of the animal, indicating that the animal was still capable of giving a galvanic skin response.

Since procaine hydrochloride in this dilution has a much greater effect on sensory end-organs and afferent nerves than it does on efferent nerves, these observations suggest that we were dealing primarily with sensory end-organs or afferent nerves.

COMMENT

These observations were interpreted as follows: 1. That the responses obtained probably are, in most cases, responses to stimulation of blood vessels and pia, rather than of the cortex itself. One might hesitate in drawing this conclusion because of the practical difficulty of stimulating blood vessels and pia without stimulating the underlying cortex. But Langworthy and Richter, as already cited,[11] working with animals under ether, found that stimulation of only a few areas of the cortex (adjacent to the motor area) gave galvanic skin responses; in our experiments these areas were avoided. To be sure, it is conceivable that under bulbocapnine and procaine hydrochloride, stimulation of other areas of the cortex might give such galvanic responses. Even if this is true and some of our responses were due to stimulation of the cortex itself, yet our results show clearly a marked difference in the frequency of response between (1) cortex, blood vessels and pia and (2) cortex and pia. In other words, even if we were stimulating the cortex itself, there is a marked difference in response when the cortex is covered by blood vessel and pia, and when that cortex is covered by pia alone. One might think of the possibility that the cortex immediately beneath the blood vessels is more responsive to stimulation than is the cortex beneath the "intermediate pia." This we consider to be highly improbable. We therefore concluded that the responses obtained probably are responses to stimulation of blood vessels and pia.

2. That the galvanic skin response in these experiments is the result of stimulation of afferent rather than of efferent nerve endings and nerves. This is a direct corollary of the first conclusion, since it is highly improbable that any efferent nerve endings or nerves would have their origin in the pia or its blood vessels, or, having their origin in the brain, would descend via the pia or blood vessels to their effector apparatus in the periphery. The observation that the application of procaine hydrochloride eliminated the responses to the areas stimulated lends support to this conclusion.

It would appear, then, from these functional studies, that faradic stimulation over the pia and its vessels sets up impulses which result in galvanic skin responses; further, that these impulses are afferent; still further, that they are primarily from blood vessels and pia rather than from the cortex itself. Moreover, these afferent impulses lead to much more frequent and larger responses when they arise from the blood

vessels rather than from "intermediate pia." It would follow from these conclusions that the pial blood vessels contain an afferent, as well as an efferent, nerve supply.

In the intact animal, such afferent impulses arising from brain vessels and perivascular spaces may well be of physiologic and pathologic importance. They would arise in regions that are probably of significance in the control of cerebral homeostasis,[13] as these regions are in the doubly advantageous position of being exposed to the influence of changes in both blood and spinal fluid. Since the cerebral circulation is in large part responsible for the maintenance of a constant internal environment for brain tissue, it would not be surprising to find that afferent impulses arise in close proximity to the vessels themselves. In other words, if there are end-organs here, they would be topographically so placed that they could play a vital rôle in the regulation of cerebral vascularity and other mechanisms which maintain the constancy of the internal environment. But this emphasis on the importance of afferent impulses arising in the proximity of blood vessels should not be taken to indicate an underestimation of the importance of such impulses arising from nonvascular pia; their functions may be of considerable significance, but do not enter into the present discussion.

To which modality these afferent impulses belong cannot be determined by these methods. Whether they arise from end-organs that in the intact animal are responsive to touch, pressure, temperature, stretch or "pain-producing" stimuli is open to conjecture. With what frequency they give rise to sensations is also a matter of conjecture. At all events, in these experiments, overt reactions commonly associated with pain were occasionally elicited. It is pertinent, also, that the galvanic skin response is most commonly associated with stimuli that produce pain or strong emotion.

It may be justifiable to point out the relationship of these observations to certain clinical speculations. These experiments offer evidence in support of the thesis that headache may result from afferent impulses originating in the cerebral blood vessels or perivascular tissue.[14] According to this thesis, alterations in the spatial relationships in these regions produce afferent impulses (the existence of which was demonstrated in these experiments), and these impulses in turn give rise to sensations of

13. A word used by Cannon to describe the general idea of uniformity or stability in the organism. Cannon, W. B.: The Sympathetic Division of the Autonomic System in Relation to Homeostasis, in The Vegetative Nervous System, Baltimore, Williams & Wilkins, 1930, p. 181.

14. Leake, C. D.; Loevenhart, S., and Muehlberger, C. W.: Dilatation of Cerebral Blood Vessels as a Factor in Headache, J. A. M. A. **88**:1076 (April 2) 1927. Wolff, H. G.: The Cerebral Circulation, in Piersol: Encyclopedia of Medicine, in press.

discomfort in the head. This thesis has been based on the observation that those drugs or situations (e. g., amyl nitrite, carbon monoxide, carbon dioxide, anemia, oxygen lack, acidosis, "asphyxia") that produce extreme dilatation of the cerebral vessels in experimental animals are those that often give a sensation of pain or fulness in the head in man.[15] The present experiments offer an explanation of this association between cerebral vasodilatation and discomfort: Evidently afferent impulses may arise from adequate stimulation on or about the cerebral vessels. Vasodilatation and its perivascular effects may provide such adequate stimulation, giving rise to afferent impulses which may in turn give rise to sensations of discomfort. It is conceivable that extreme vasoconstriction also may provide adequate stimulation for the production of such impulses, with resulting headache, but for this there is no satisfactory evidence.

In a similar way, these experiments add evidence in support of one hypothesis of the mechanism of convulsions associated with intracranial scar formation or tumor growth. Penfield and Foerster,[16] in a discussion of traumatic epilepsy, stated that "The blood vessels form in one sense the woof of the contracting network [of a scar]. Traction, therefore, upon the vessels must be inevitable. The hypothesis at once suggests itself that a vasomotor reflex secondary to this traction is responsible for the initiation of the convulsive seizures." Such a hypothesis implies that there are afferent impulses taking part in this vasomotor reflex; the experiments here described offer evidence for their existence. To restate the hypothesis, then, in terms of the new data: If the blood vessels or adjacent pia are stimulated by a change in spatial relationships due to the contraction of a scar, afferent impulses may be set up and reflexly produce vasomotor effects in the brain vessels. These vasomotor changes may seriously alter the cerebral blood supply, resulting in circumscribed circulatory deficiency and subsequent convulsions.

SUMMARY

1. Bulbocapnine administration provided an adequate method by which cats under local anesthesia could be immobilized without interfering with the galvanic skin responses.

2. Galvanic skin responses were obtained by faradic stimulation on the pial blood vessels and on the relatively nonvascular pia over the cerebral cortex.

3. Stimulation on the pial vessels produced galvanic skin responses more than twice as often as did stimulation on the relatively nonvascular

15. Wolff (footnote 14).

16. Penfield, W., and Foerster, O.: The Structural Basis of Traumatic Epilepsy and Results of Radical Operation, Brain **55**:99 (July) 1930.

pia. Further, stimulation on the vessels produced galvanic skin responses which were much larger than those that resulted from stimulation on the relatively nonvascular pia.

4. It was concluded: (*a*) that the impulses produced by the stimulation were afferent rather than efferent; (*b*) that these afferent impulses had arisen in blood vessels and pia rather than in the underlying cortex; (*c*) that the responses had more than twice the frequency and magnitude when the afferent impulses arose from blood vessels than when they arose from the relatively nonvascular pia, and therefore (*d*) that the pial blood vessels possess an afferent, as well as an efferent, nerve supply.

5. Afferent impulses from the pial blood vessels may be of importance physiologically in the mechanisms involved in the maintenance of a constant internal environment through the regulation of the cerebral circulation, as well as pathologically in the production of certain headaches and convulsions.

CONCLUSION

It is probable that the nerves on the pial blood vessels convey afferent as well as efferent impulses. In addition to their physiologic significance, such afferent impulses may be of importance in the genesis of certain headaches and convulsions.

CHRONIC SYPHILITIC POLIOMYELITIS

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The idea that syphilis may be the etiologic factor in the production of wasting of muscles is by no means new. As far back as 1795, Graves [1] was led to ascribe to syphilis an atrophy that disappeared under antisyphilitic remedies. In his textbook, Gowers [2] (1896) stated that chronic muscular atrophy "sometimes succeeds syphilis, and no other cause may be traceable."

It was, however, not until Raymond [3] that a microscopic study was made of the spinal cord from a patient in whom there was a degeneration of the anterior horn cells as well as a diffuse meningomyelitis.

Léri,[4] in 1903, in a paper entitled "Atrophies musculaires progressives spinales et syphilis," which he read before the Congress of Alienists and Neurologists, described six cases of muscular atrophy, all of which were in syphilitic patients. Two autopsies showed the lesions of a diffuse vascular meningomyelitis analogous to that observed by Raymond.

In 1922, Léri [5] again discussed the question and stated the belief that progressive amyotrophy due to syphilis was not the exception but the rule, stating that "la syphilis est la cause de beaucoup la plus frequent des lésions spinales qui determinent les amyotrophies progressives." (Syphilis is the most frequent cause of lesions of the spinal cord that result in progressive amyotrophy.)

From the clinical side, Léri established that: (1) as a rule, the atrophy shows inequality on the two sides; (2) most of the cases show the "atonic" type of atrophy—atrophy with loss of reflexes in the wasting muscles, and (3) the absence of the classic clinical features of syphilis is the rule.

From the Laboratory of Neuropathology of the Philadelphia General Hospital and Temple University Medical School.

Dr. Charles W. Burr, in whose service this patient was and through whom a good history and physical examination were obtained, gave me permission to use this case.

1. Graves, R. J.: Clinical Lectures on the Practice of Medicine, Dublin, Fannin & Co., 1849, vol. 1, p. 509.

2. Gowers, W. R.: Diseases of the Nervous System, Philadelphia, P. Blakiston Son & Co., 1903, vol. 1.

3. Raymond: Semaine méd. **13**:56, 1893.

4. Léri: Atrophies musculaires progressives spinales et syphilis, Congrès des aliénistes et neurologistes, Bruxelles, 1903.

5. Léri: Les atrophies musculaires syphilitiques, in Questions neurologiques d'actualité, Paris, Masson & Cie, 1922, p. 259.

From the pathologic side, Léri believed that the disease was not a systematic anterior poliomyelitis but a diffuse vascular meningo-myelitis. He found the essential lesion of the cells of the anterior horns to be a pigmentary atrophy, with a meningeal infiltration with lymphocytes, which forms, especially in the region of the anterior roots, veritable nodules that resemble gummas. The vessels entering the cord are surrounded by lymphocytes and may show syphilitic changes. The cord itself may show changes in its posterior tracts resembling tabes, or in the pyramidal tracts, resembling amyotrophic lateral sclerosis.

Léri in this article referred to the cases of atrophy limited to the small muscles of the hands reported by Marie and Foix in 1912. The latter described the pathologic process in this condition as the result of syphilitic changes in the vessels with consequent softening in the anterior horns (tephromalacia) at the level of the seventh and eighth cervical segments.

Martin,[6] in 1925, under the term "amyotrophic meningomyelitis," emphasized again the causal relationship of syphilis to the atonic type of atrophy. He reported one case with the necropsy study, in which the history and physical symptoms were similar to those of amyotrophic lateral sclerosis, but with positive serologic reactions. Microscopic study of the spinal cord showed a diffuse degeneration of the white tracts, posterior, anterolateral and marginal, as well as a cellular degeneration in the anterior horns and chronic leptomeningitis throughout the whole length of the cord and medulla. A moderate degree of arteritis affecting chiefly the internal and adventitial coats of the vessels was present.

Martin also examined the sections in a case reported by Williamson in 1901. This patient resembled the one whose case is reported herewith to a remarkable degree. He, too, had atrophy of the muscles of the upper limbs, shoulder girdle and neck. "For 20 years before his death his arms had hung helpless by his sides and his head was bent forwards." No signs of bulbar paralysis were present. Microscopically, in the cervical cord "not a single anterior horn cell could be found." The horns were shrunken and glia was increased. Weigert sections revealed a marginal degeneration with a mild pyramidal tract degeneration on one side. The evidence given for suspecting the condition to be syphilitic was not convincing.

REPORT OF CASE

History.—J. N. a white man, aged 49, was admitted to the Philadelphia General Hospital on July 4, 1929, and died on Oct. 5, 1929. His complaints on admission were: inability to hold the head erect and difficulty in breathing and swallow-

6. Martin: Amyotrophic Meningo-Myelitis (Spinal Progressive Muscular Atrophy of Syphilitic Origin), Brain **48**:153, 1925.

ing except when the chin was held up. According to the patient, the trouble had begun about December, 1927, with difficulty in breathing and in phonation. Four months later, he began to note wasting and weakness of the muscles of the hands. This gradually progressed and ascended slowly, so that at the time of admission the forearms, arms and shoulder girdles were greatly wasted and practically useless. To flex the forearm he had to swing it and actually throw it up. Gradually, within the past year there had developed atrophy of the muscles of the neck, so that there had been more and more difficulty in holding the head erect, until when he was admitted to the hospital it would fall either forward until the chin rested on the chest or backward. He was without power to move the head by the muscles of the neck alone. In eating he had to support the chin in order to be able to swallow.

From the previous history, it was learned that he had had a chancre twenty-two years before. He had received practically no treatment. In 1919, he was injured, the back of the head being mainly involved; the scalp was cut, and he was unconscious for an unknown period.

Examination.—The patient was tall and thin, and his head could be said to be loose on the shoulders. For the most part he sat with the arms supported on a table and the chin in his hands. The pupils were small; the right was sluggish, the left fixed. The cranial nerves were normal, and the twelfth nerve particularly was intact. There was intense atrophy of the muscles of the neck, so that the cervical vertebrae were easily palpable. Fibrillary twitchings were present in the neck muscles.

The upper limbs likewise showed atrophy, not only in the intrinsic muscles of the hands, but in the forearms and shoulder girdles. Fibrillary tremors were very marked. Vasomotor disturbances were evident. The reflexes of the upper limbs were absent.

In the lower limbs there was practically no atrophy, and the reflexes at the knee were slightly hyperactive, with the reflexes at the ankle absent. No abnormal reflexes were discovered.

Careful examination for sensation of all forms revealed it to be normal in all respects. Because of a suggestion that the case might be one of syringomyelia, pain and temperature sensations were carefully tested and found intact.

From the laboratory standpoint the only important features were 4+ Wassermann reactions of the blood and spinal fluid, with a colloidal gold curve of 4443322100.

Diagnosis.—Because of the involvement of the motor divisions of the upper cervical nerves, with atrophy and fibrillations, syringobulbia was considered only until it was determined definitely that the sensory sphere was intact. A diagnosis was made therefore of chronic anterior poliomyelitis of syphilitic origin.

Course.—Antisyphilitic treatment was instituted, but little good was accomplished because of the rapidity with which the lesion had progressed. About four months after admission, the patient died of a complicating lung condition.

During his stay in the hospital, physical examination of the chest and abdomen gave negative results until toward the end, when pneumonia developed, the result probably of aspiration of food into the lungs. The atrophy of the muscles around the neck and shoulder became so intense that the deep bony landmarks could actually be seen without difficulty. Atrophy of the hands also progressed, but to a lesser degree than that of the shoulder girdle.

Pathologic Examination.—Spinal Cord: Weigert-Pal sections showed a normal myelin sheath picture of the white fiber tracts. On close inspection it was seen

that there was a poverty of medullated fibers in the gray matter, especially in the anterior horns, and that the horns appeared shrunken. This was especially marked in the entire cervical region (fig. 1) and in the upper thoracic region, but decreased as the cord descended, until in the lumbar region the fibrillary make-up of the entire horn was practically normal.

It was, however, with the toluidine blue stains of alcohol-fixed material that the basic pathologic process was disclosed.

Meninges: There was a marked meningeal infiltration of round cells, most of which were lymphocytes; there were also some plasma cells. This infiltration was particularly marked in the anterior fissure and apparently was in direct communication with the process within the cord substance, since the perivascular infiltration

Fig. 1.—Cervical cord, showing shrinkage of anterior horns and poverty of medullated fibers. Myelin sheath stain.

was continued from the vessels of the cord substance to the subarachnoid space. There was a mild reactive fibrosis of the soft meninges.

Blood Vessels of the Spinal Cord: The large blood vessels showed a mild thinning and fibrosis of their walls. The small vessels for the most part showed marked thickening of the walls with hyalinization. There was definite swelling of the lining cells in some of the small vessels.

Histology of the Spinal Cord: The cord showed the most marked changes in the anterior horns, a process that at first glance, especially in the cervical region, gave one the impression of being extremely similar to that seen in acute anterior poliomyelitis (fig. 2). There was, as in the latter condition, a marked reduction in the number of ganglion cells, while the vessels showed perivascular collaring with

round cells that were both lymphocytic and glial, especially the former. There was not, as in acute poliomyelitis, congestion and dilatation of the blood vessels. Hemorrhages were likewise entirely absent. Unlike the acute condition, there was much less diffusion of the perivascular infiltration into the tissue. Finally, there was no neuronophagia, a fairly constant condition in acute poliomyelitis. The ganglion cells in the anterior horns that remained did not show the lipoid degeneration that is seen particularly in progressive spinal muscular atrophy. Nor did they show the axonal chromatolysis that is seen in the anterior horn cells in atrophies that occur in multiple neuritis. There was, on the other hand, simple cell shrinkage (einfache Schrumpfung) as the most common change in the cells, with here and there sclerosis of the cells (Sklerose of Nissl).

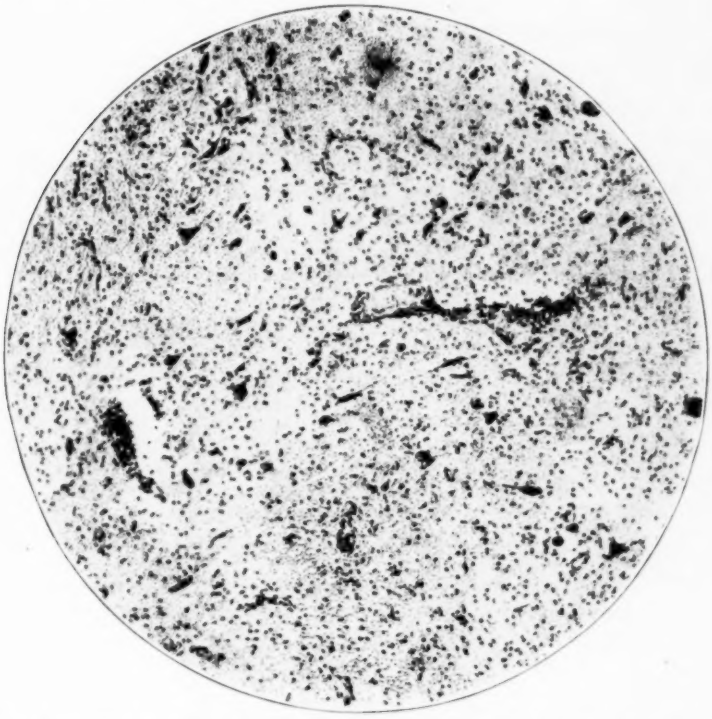

Fig. 2.—Anterior horn, showing severe ganglion cell disease and cell poverty in midst of a chronic inflammation. Toluidine blue stain.

In the white matter one noted no actual degeneration of tissue, but there were present small vessels with occasional groups of lymphocytic cells in the perivascular spaces, which in the marginal vessels seemed to communicate with the subarachnoid space and with the vessels in the anterior horn.

The anterior and posterior roots showed a mild, yet definite, infiltration with lymphocytes, most of which was in direct communication with the meningeal infiltration.

Cerebrum: The meninges showed marked edema, with a mild infiltration of round cells of the lymphocytic type and an occasional plasma cell. An increase in fibroblastic tissue was present.

The large blood vessels showed both atherosclerosis and fibrosis. The small vessels showed swelling and proliferation of the lining cells, with thickening and hyalinization of the media.

The cortex itself showed little change; the lamination was normal, and glia was not in great excess. The ganglion cells showed a mild chromatolysis, with occasional ischemic cell change, and there was probably a mild cellular decrease. Occasionally a perivascularly infiltrated blood vessel was to be seen.

The subcortex showed hyalinized small blood vessels and occasional perivascular collections of phagocytic cells, many of which still contained pigment—evidence of a previous degenerative change, a not uncommon condition in older people.

Medulla: The lower part of the medulla showed the same type of change that was seen in the upper part of the spinal cord. There were the same character and degree of infiltration of the meninges, especially in the anterior half of the medulla. There was an occasional vessel within the medullary substance that showed a marked perivascular infiltration, especially in the floor of the fourth ventricle. The small vessels showed the same type of swelling of the lining cells of the medium and small-sized vessels.

The motor nuclei within the medulla, while not showing the intense change seen in the ventral horns of the cervical cord, showed changes of a lighter degree. The sensory nuclei were not involved.

Cerebellum: There was a definite, yet mild, infiltration of the meninges with round cells of the lymphocytic type. There was no marked decrease in Purkinje cells. The dentate nucleus showed a moderate degree of atrophy of its ganglion cells. Within it was seen an occasional vessel showing perivascular infiltration.

COMMENT

There is described a case of muscular atrophy of severe degree, atypical in its distribution, in which the etiologic factor was found to be syphilis, both clinically and pathologically. The rôle of syphilis in the production of muscular atrophy has been stressed so much by French writers that, at times, in reading their literature one obtains the impression that all cases of atrophy have a syphilitic etiology. That this is not true is amply proved by the many cases of progressive muscular atrophy, amyotrophic lateral sclerosis and true bulbar palsy in which syphilis is not present serologically or at autopsy. It is probably true that Gowers' interpretation of conditions of this sort is correct. Gowers coined the word "abiotrophy," by which he meant the premature decay of a particular structure. No one today explains a premature cataract on a syphilitic basis. Other forms of abiotrophy are seen in premature arteriosclerosis and similar conditions that may terminate life. It is well known that familial arteriosclerosis can occur without the presence of syphilis. It is just as possible to have a premature death of the motor cells in the central nervous system, which forms one unit, as it is of any other unit structure in the body. Moreover, pathologically, in the examination of the spinal cord and brain stem, in cases of atrophy, a simple shrinkage, atrophy or lipoid pigmentation in the motor cells is found. The occasional presence of a

mild round cell infiltration in the meninges in cases of this sort does not spell syphilis, but rather suggests degeneration.

It is therefore not in the typical cases of atrophic conditions that syphilis usually plays a rôle, but rather in the atypical, and it is with an atypical case that this paper is concerned. Whenever the clinical picture does not fit in with the accepted symptomatology of the various forms of atrophy, syphilis must be considered. In the patient whose case is reported here, the atrophic manifestations were most marked in the muscles of the neck, and to such a degree that it prevented the patient from holding his head erect. The upper limbs were affected much less. An occasional case of this sort has been reported in the literature. Spiller,[7] in 1912, recorded the case of a man, aged 61, who said that he had had a syphilitic infection. This patient was unable to hold his head erect because of atrophy of the muscles of the neck.

> It falls forward on his breast or backward. The muscles of the hands, upper limbs, shoulder girdle and neck were greatly atrophied. Fibrillary tremors are present below the clavicles and in the forearms. Voluntary power in the upper limbs is feeble. He can not abduct the upper limbs to a right angle with the body, or raise them above his head, but he can flex the forearms on the arms and flex and extend the wrists. He can not flex or extend either thumb. The first and ring fingers of the left hand can be extended, the others very little. The grip in each hand is feeble. The biceps tendon reflexes are diminished, the triceps tendon reflexes are a little increased.

From the facts given in Spiller's case, one notes that it is absolutely identical with that of the patient presented in this paper. Unfortunately, no autopsy was obtained in Spiller's case.

Recently, there was admitted to the Philadelphia General Hospital another patient whom the staff recognized immediately as a counterpart of the patient whose case is presented in this paper and with whose blood a positive Wassermann reaction was obtained. Fortunately for the patient, he came to the hospital much earlier in the course of the disease and he was given energetic antisyphilitic treatment; he has made practically a complete recovery.

The literature has been confusing as to the rôle that syphilis plays in the etiology of conditions of this sort, because the pathologist has taken a mild round cell infiltration in the meninges as *prima facie* evidence that syphilis is the etiologic factor. Cases of this sort have been reported, among others by Leopold,[8] in which the clinical picture is practically a textbook picture of amyotrophic lateral sclerosis.

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7. Spiller: Syphilis a Possible Cause of Systemic Degeneration of the Motor Tract (case 4), J. Nerv. & Ment. Dis. **39**:584 (Aug.) 1912.

8. Leopold: A Case of Progressive Muscular Atrophy with Necropsy, Probably Syphilitic in Origin, J. Nerv. & Ment. Dis. **39**:9 (Sept.) 1912.

Leopold found intense involvement of the ganglion cells in the anterior horns, with marked reduction in their numbers, and chromatolysis and pigmentation in the remaining cells. The medullary sheath stains conformed to the textbook picture. The only statement in the entire pathologic description on which a diagnosis of syphilis was made is as follows: "And around some of the vessels a moderate round cell infiltration is noted. It is probably indicative of syphilis." This very moderate cellular infiltration occurred practically entirely in the meninges. To make a diagnosis of syphilis on evidence so slim is not justified, and today syphilis would not be considered in a case of this sort. The vessels themselves showed no evidence of syphilitic involvement. This is but one of the many examples in the literature in which on insufficient grounds syphilis has been considered the basic factor.

From the clinical standpoint, one may quote Martin [9] who stated:

> The atrophy in amyotrophic syphilitic meningo-myelitis is of the atonic type; it begins with great constancy in one of three situations, it is frequently unilateral, the lower limbs often become spastic, bulbar symptoms are uncommon, severe pain may occur at the onset of the wasting, the vibration sense may be impaired, there may be difficulties of sphincter control. Such characters should, of themselves, suggest syphilis and lead to the examination of the blood and cerebrospinal fluid.

It has been my experience that only in atypical cases does syphilis play a rôle, and the few cases that have come under my observation in which syphilis has been etiologic have been so atypical that one could immediately recognize them. I have already reported the case of a patient in whom atrophy of one hand occurred. Here, a positive serologic study determined the therapeutic procedure, and the patient made a complete recovery under antisyphilitic remedies. It is possible, although not seen by me personally, to have a typical form of atrophy in a patient who has also had a syphilitic infection, but in patients of this sort the clinical response, or rather lack of response, will soon determine that the syphilitic infection has nothing to do with the atrophy. It is, of course, evident that when a condition is advanced and the cells of the anterior horns are destroyed, even if the disease is due to syphilitic involvement therapy will have no effect, so that the results of therapy cannot alway be used as a criterion for a syphilitic etiology. In the case reported in this paper vigorous treatment was instituted as soon as it was definitely determined that syphilis was a factor, but the patient's condition was too far advanced, and no improvement was noted.

SUMMARY

1. A case of atypical progressive spinal muscular atrophy is reported.

9. Martin (footnote 6, p. 179).

2. Serologic tests strongly indicated a syphilitic infection.

3. Treatment was of little value because of the advanced clinical picture.

4. Pathologic study showed a chronic inflammatory and degenerative process in the upper part of the spinal cord and lower part of the brain stem, which had all the appearances of a syphilitic involvement.

5. Atypical cases of the atrophies suggest a syphilitic etiology, while typical cases are usually nonsyphilitic. Early treatment must be instituted in the syphilitic variety in order to secure clinical improvement.

Clinical Notes

ABSENCE OF OCCIPITAL LOBE OF BRAIN (PORENCEPHALY) WITH ESSENTIALLY NORMAL VISION

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Porencephalic cysts of the brain are of sufficiently common occurrence to preclude the publication of an isolated case unless it presents some unusual features of clinicopathologic significance. The case to be reported is of special interest because the findings shed some light on the problem of cerebral function and localization, especially as regards the visual centers and pathways.

Porencephaly was first described by Heschl [1] in 1859, with a detailed report of eight cases. He believed the condition always to be associated with hydrocephalus, "the delicate brain substance or primitive cerebral wall being affected by increased intraventricular pressure, especially at growing points."

Since this description, many clinical and pathologic reports have appeared in the literature.[2] The most comprehensive discussion of recent years appeared in 1925, by LeCount and Semerak.[3] These authors reviewed in detail the pertinent literature as regards the incidence, etiologic factors and anatomic location of the defect. From a study of four cases of porencephaly they concluded that the defect is usually congenital in origin, but may be acquired, and that it results from vascular obstruction of either traumatic or inflammatory origin. Most authors believe that the defect is primarily due to interference with the vascular supply to the part and, as Holtby [4] pointed out, the cyst usually occurs in the areas supplied by either the anterior, middle or posterior cerebral arteries.

It is generally agreed that the term porencephaly should be reserved for defects in the brain substance that communicate with the ventricular system and are covered on the outside by the arachnoid. Clinical symptoms depend on the site and extent of the lesion.

From the Departments of Surgery and Psychology, University of Louisville.

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3. LeCount, E. R., and Semerak, C. B.: Porencephaly, Arch. Neurol. & Psychiat. **14**:365 (Sept.) 1925.

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REPORT OF A CASE

Porencephaly, left occipital lobe; birth trauma; focal epilepsy; normal vision; encephalogram; operation; recovery.

Clinical History.—A. H., a white boy, aged 11 years, was referred by Dr. Frederick Speidel of Louisville, Ky. On Dec. 15, 1930, when first seen, he seemed quite alert, cheerful, talkative and, from a casual observation, normal.

The patient was a second child, delivered by forceps after a long labor. A soft area in the occipital region (the side is not remembered) was noted at birth by the family physician and was of sufficient size to give him considerable concern for the first month. Perhaps undue significance was attributed to this lesion at the time, because convulsions occurred shortly after birth and continued at varying intervals for the first six weeks. During this time, the soft spot or defect in the occipital region steadily receded, and in a few more weeks completely disappeared. Occasional frontal and bitemporal headaches continued to occur at irregular intervals. The patient had the usual diseases of childhood and pneumonia without complications. Otherwise, the developmental history was normal. He is normally lefthanded, although he writes with the right hand, owing to training. He eats, grasps, pitches, tosses, bats and digs with the left hand.

In February, 1930, mild epileptiform seizures again occurred. These spells consisted merely of a loss of volitional motor power with increased tone of the extremities. There were no clonic movements, frothing of the mouth or biting of the tongue. He always was able to recognize the onset of an attack, and he never fell or injured himself. On one occasion there was loss of sphincteric control, and the right side of the body became temporarily numbed. These seizures occurred about every two weeks and lasted for from twenty to thirty minutes each time. Sleep usually followed an attack.

Neurologic examination gave normal results in all respects. Ophthalmoscopic examination revealed normal disks and fundi. The perimetric fields were normal except for slight constriction of the right half of the field of vision of each eye.

Encephalographic studies were made on Jan. 9, 1931. Colonic ether anesthesia was used. The spinal fluid pressure was normal. Owing to a rather alarming reaction to the injection of air, all of the cerebrospinal fluid was not replaced with air. The encephalogram revealed an absence of cortical markings in the left occipitoparietal region. This area, easily identified on both anterior, posterior and left lateral stereoscopic films, was less opaque to the x-rays than the surrounding brain tissue. The left lateral ventricle, fairly well filled with air, ended abruptly at this area. The right ventricle appeared to be normal. No other abnormal cortical markings were found on either side (fig. 1). Roentgen-ray Diagnosis: Probable porencephalic cyst of the left occipital lobe.

Examination of the blood and urinalysis gave normal findings. The Wassermann reaction of the blood and spinal fluid was negative. A chemical analysis and cell count of the spinal fluid gave normal results.

Preoperative Diagnosis.—Epilepsy; probable porencephaly of the left occipital lobe.

Operation.—On Jan. 9, 1931, a left occipitoparietal bone flap was turned down in the usual manner. The exposed dura was bulging, and a large collection of fluid lay beneath it. When the dura was opened, a profuse amount of clear fluid escaped through the incision. Following the loss of fluid, the posterior part of the exposed dura collapsed. The dural flap was reflected with its base

toward the anterior portion of the wound. The dura was thickened, and what appeared to be arachnoid was densely adherent to it. The remainder of the fluid was evacuated by suction. No remnant of cerebral substance remained in the area normally occupied by the occipital lobe. This fluid-filled space was bounded anteriorly by cerebral tissue which had the appearance of a cross-section at about the junction of the parietal and occipital lobes; the relationship of the gray and white matter was clearly defined, and the openings into the body and temporal horn of the lateral ventricle were visible. The choroid plexus lay on the floor of the ventricle. The midbrain and the tip of the thalamus were in plain view. A tuft of choroid plexus, about 1 cm. long, extended posteriorly from the open ventricle and floated in the fluid-filled space. This portion of the plexus was excised for microscopic study. Mesially, the defect was bounded

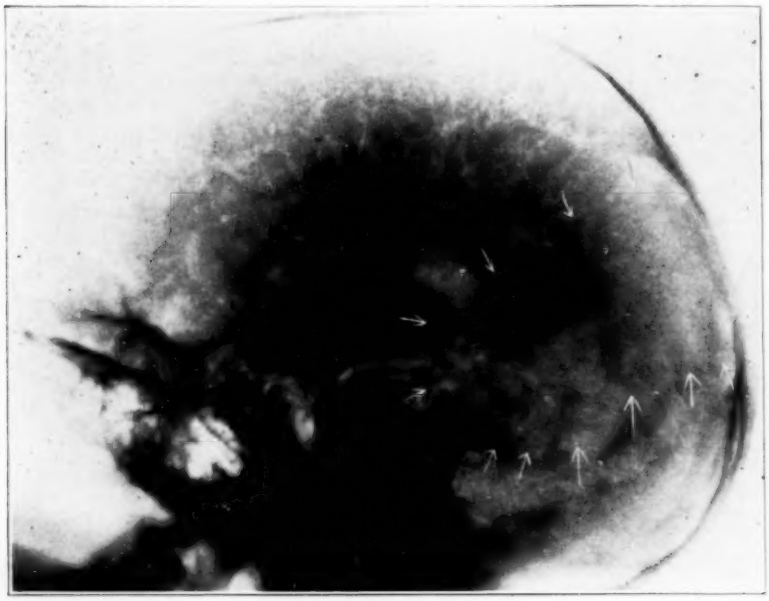

Fig. 1.—Encephalogram, lateral view, with right side of head nearest to film, showing incomplete replacement of fluid with air. Note the absence of cortical markings in the left occipital region (outlined with arrows). Outline of the left lateral ventricle ends abruptly at this area. When films are studied stereoscopically, the abnormal markings in the left occipital region are more clearly defined.

by the falx cerebri; posteriorly and inferiorly, by the tentorium. The outer margin of the parietal lobe was densely adherent to the dura by what appeared to be degenerated cortical and fibrous tissue. After all the fluid was removed and the choroid plexus was dried with cotton pledgets, a small pool of clear, apparently normal cerebrospinal fluid formed about it promptly. Production of fluid by the plexus was demonstrated repeatedly by this method. With the exception of the portion attached to the dura as already described, the cerebral tissue of the parietal lobe was normal in appearance (fig. 2). The adhesions between the dura mater and the parietal lobe cortex were freed, and the abnor-

mal tissue was removed, leaving no attachments of the cortical tissue to the dura mater. The cavity was filled with physiologic solution of sodium chloride, and the dura was sutured tightly. Closure was made in layers with black silk.

Microscopic sections of the excised choroid plexus showed the usual intricate papillary portions and blood vessels, generally distorted by an old scarring fibrosis. The walls of the blood vessels were moderately thickened. There were numerous areas of calcareous deposits and of degeneration with hyalinization. Microscopic Diagnosis: Chronic inflammation with fibrosis and calcareous infiltration.

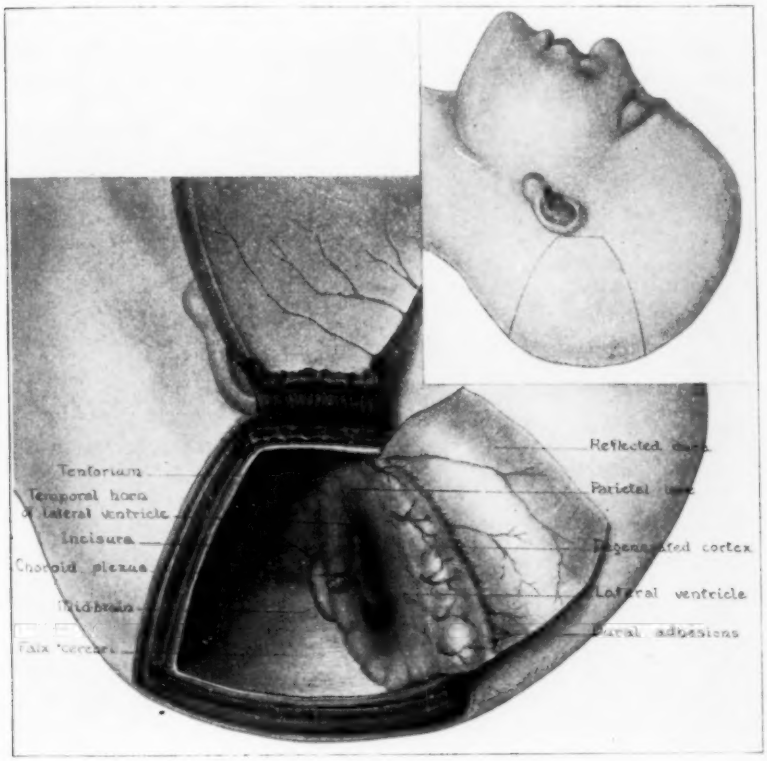

Fig. 2.—Drawing showing porencephalic defect in the occipitoparietal portion of the left hemisphere as disclosed at operation. Insert shows position of the osteoplastic flap.

Course.—Convalescence was uneventful, except for a rather persistent febrile reaction lasting for about fifteen days. The general condition of the patient was good throughout the convalescence. At no time was there riding of the bone flap. The patient was discharged from the hospital on the sixteenth postoperative day free from symptoms.

Since the operation, the patient has been entirely free from headache, and there has been no recurrence of the epileptiform seizures. The visual field studies are reported in detail:

Postoperative Visual Examination.—On Feb. 7, 1931, less than a month after the operation, a careful determination was made of the patient's visual functions, including the extent of the visual fields, absolute and relative scotomas, zonal sensitivity to color and binocular parallax.

The visual fields were tested on a large black tangent campimeter. Fixation was monocular at 1 meter with the unused eye completely blindfolded. The campimeter was homogeneously but not strongly illuminated over its entire area of 3 by 2.5 meters. The intensity of illumination was such that for the eyes of the examiner, which are normal, the field for the left eye coincided approximately with the field for the left eye of the patient, and that of the examiner's right eye was symmetrical with that of the left (as the patient's was not).

The patient was first tested on eight meridians for each eye with white stimulus objects, 10 mm. and 1 mm. in diameter. The observations were repeated

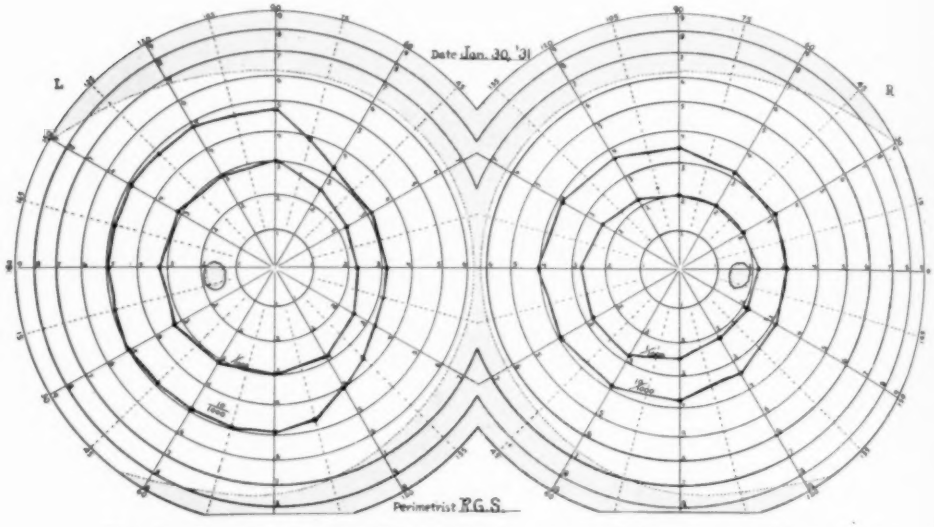

Fig. 3.—Outline of visual fields with 10 mm. and 1 mm. test objects at a distance of 1,000 mm. from the campimeter. Note the moderate homonymous constriction of the right field.

five times in succession on each meridian, and then those on the right half of each field (corresponding to the missing cortical tissue) were repeated five times again in order to insure the reliability of the determinations in the anomalous areas. The patient was extremely cooperative and intelligent, maintaining fixation and avoiding movements of the eye with surprising constancy. The results are given in figure 3. In the field of vision of the left eye, the nasal half (corresponding to the absent cortical tissue) is somewhat smaller than the nasal half (normal) of the field of the right eye. In the field of vision of the right eye, the temporal half (corresponding to absent cortical tissue) is somewhat smaller than the temporal (normal) half of the field of the left eye. Thus is appears that each half which was deprived of its cortical projection in the left occipital lobe did not succeed in establishing a fully equivalent vicarious projection area. But even with this condition, it is evident that there is for neither eye the loss of vision that would correspond with the absence of occipital tissue.

An exploration of the nasal half of the left field and of the temporal half of the right field failed to reveal abnormal blind spots which might conceivably correspond to the absent tissue. Nowhere was there an area of absolute scotoma with a 10 mm. stimulus object, or of relative scotoma with a 1 mm. stimulus object. Vision was clear and unequivocal throughout both fields, with the exception of Marriotte's blind spots, which were normal in situation and extent.

A test was made also of zonal sensitivity of the retinas to color, i. e., of the changes that a constant stimulus produces in passing from periphery to fovea. Here, too, the results coincided with those in the normal eye. A red stimulus object of 10 mm., when brought in on any meridian from the periphery toward the center, underwent the changes characteristic of the normal eye. It first became visible as black, then yellow, then orange and finally, at the fovea, red. Analogous results were obtained with green, blue and yellow stimulus objects.

To this point, all the tests were monocular and directly significant. While tests of binocular function do not have as immediate a significance in this case as those of monocular function, it was advisable to learn as much as possible concerning the visual behavior. Consequently, the parallax was observed in the Bourdon apparatus, consisting of three vertical threads the middle one of which is equidistant from the other two and may be shifted in and out of the plane of the others. The patient made exact reports of the position of the middle thread in relation to the plane of the other two. His results were somewhat better than those of several average observers who were tested under the same conditions in order to establish a norm. In order to make sure that he was not relying on unsuspected secondary depth cues, he was tested monocularly with the same apparatus, and here at once the errors of his judgment increased tremendously. This showed definitely that as regards parallax he possessed normal binocularity. Further tests with the binocular and monocular localization of a suspended needlepoint in space, of fusion of stereoscopic pictures (especially constructed to be unrecognizable unless fused), of binocular retinal rivalry of colored fields and of binocular color fusion demonstrated the presence of normal binocularity. A simple acuity test showed normal vision for both eyes.

COMMENT

It would be highly desirable to know when and how the porencephalic defect in the left hemisphere occurred. There is some indirect evidence, especially the history of a "soft spot" in the occipital region after a difficult instrumental labor and convulsive seizures for the first six weeks of life, which would point to birth trauma as being the exciting agent. The observation of the parents that the "soft spot" disappeared after a few weeks with a cessation of the convulsions also may have some significance. The operative findings of dense adhesions between the meninges and cortical substance would be suggestive of an old traumatic or inflammatory lesion.

If birth trauma is the causative agent in this case, one may assume either that the blood supply to the part was suddenly cut off by direct trauma to the arterial supply or that a large hemorrhage occurred in the cerebral substance, which, in turn, compressed the posterior cerebral artery. Unfortunately, the history and findings are not sufficiently complete to justify the assumption that trauma at birth was responsible for the production of the cerebral defect.

If the defect was produced during intra-uterine life, it may have resulted from either vascular occlusion or an arrest in development of the left cerebral hemisphere. The defect corresponds fairly accurately with that part of the

hemisphere supplied by the posterior cerebral artery. Assuming that occlusion of this vessel occurred before birth and that cerebral substance disintegrated and became absorbed, it still must remain a matter of conjecture as to whether the visual centers and optic radiations had developed at the time of the vascular accident.

No evidence was obtained in this case to shed light on the question of developmental arrest being the causative factor in the production of porencephalic defects.

This patient possesses, except for moderate constriction of the right visual field, normal monocular and binocular vision in the absence of a normally placed cortical mechanism for the left half of each retina. This is most surprising in view of the fact that there is abundant evidence in the literature to show that when the visual centers are destroyed in adult life permanent defects are produced in the visual fields corresponding to the visual areas destroyed.

Two possible explanations for this apparent discrepancy should have careful consideration: First, the visual cortex was not destroyed but merely displaced forward, due to a cystic tumor, an inflammatory cyst or a hematoma. Second, there was destruction of the cortical visual centers by factors concerned in the production of porencephalic defects, with the establishment of a vicarious projection center in another portion of the brain.

Displacement of the occipital lobe by a cyst due to tumor, inflammation or hemorrhage obviously would predicate an increase in the quantity of intracranial contents. With an increase of cranial contents, intracranial pressure would be elevated. If the fluid-filled space in this case had been due to a cyst which had displaced the occipital lobe anteriorly, there should have been a marked increase of intracranial pressure. The only symptom suggesting an increased pressure was headache. No signs of increased intracranial pressure were present; there was no papilledema or bradycardia, and the spinal fluid pressure was normal. Furthermore, a cyst of the type under discussion should possess a definite wall. No cyst wall other than the meninges was demonstrated in this case. Cystic fluid is characteristically yellow and clots on being exposed to the air. The fluid in this case was clear, resembling normal spinal fluid. Cysts of the aforementioned varieties do not communicate ordinarily with the ventricular system; in this case, there was free communication between the ventricle and the fluid-filled space. Lastly, had the visual centers been displaced anteriorly, the compressed cortical tissue should have occupied the anterior part of the defect. The anterior boundary of the defect, however, consisted of a cross section of cerebrum with landmarks that were unmistakable, gray and white matter clearly defined, the open temporal horn and body of the lateral ventricle with choroid plexus on the floor of each.

That the occipital lobe of the left cerebrum was destroyed in the production of the porencephalic defect seems more reasonable in view of the operative findings. In the use of the term porencephaly one presupposes absence of brain substance in the area involved. Whether this destruction occurred before the visual cortex and optic radiations were differentiated is unknown. In either event, one must assume that a vicariously placed visual center had developed, since the patient possessed normal visual characteristics in a slightly constricted right visual field. If the destruction of the left occipital lobe occurred before the visual center became differentiated, the establishment of a new center elsewhere with proper connecting pathways would seem less difficult of accomplishment than the formation of a new visual mechanism after a well differentiated one had been destroyed. Furthermore, if these destructive processes occurred after myelinization and after the establishment of function by the visual apparatus,

including numerous association and connecting pathways, a far more complicated developmental process would be required to produce a new center of vision.

Assuming that a vicarious projection center has been established elsewhere in the cortex to assume the functions of the absent lobe, it becomes an interesting speculation as to where such a center might be located. The first possibility that suggests itself is that the fibers from the entire retina pass to the right occipital lobe, and that the visual projection area is enlarged to compensate for the absence of the left. There is some argument in favor of this view in the patient's complete and pronounced left-handedness. In fact, any one conversant with the results of Papez [5] and Dallenbach [6] might have predicted the patient's left-handedness from the position of the lesion. Another possibility would be that a new cortical center has been established in some part of the normal portion of the left hemisphere. Unfortunately, there are no means at our disposal by which such a new center for the right visual field can be located. It would seem, therefore, that the patient has a cortical visual center which is functioning for the absent one, and the suggestion is offered that the new center is either in the right occipital lobe or in the normal portion of the left cerebrum.

404 Brown Building.

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MALIGNANT NEUROFIBROMA OF THE SCALP

Report of a Case

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AND

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We wish to report a case in which a congenital tumor of the scalp grew to a tremendous size, hung behind the right ear and had racemose extensions over the parietal and temporal regions and into the right orbit.

REPORT OF CASE

History.—In 1926, a boy, aged 13, was brought to the Mayo Clinic by his mother, who said that when he was 8 months of age she first had noticed two small lumps, "the size of peas," behind his right ear. By the time he was 5, these lumps had grown, had coalesced and had extended over the right ear to the right temporal region. When he was 9, the tumor had become still larger, but most of the growth had been in the right temporal region, making this the more conspicuous part.

From the Mayo Foundation and the Section on Neurologic Surgery, the Mayo Clinic.

During the year before he came to the clinic, the tumor had grown very rapidly, especially the posterior part.

Examination.—The tumor was soft, rounded and flattened against the skull, and there were multiple subcutaneous nodules extending over the right ear into the right orbit. The ear was depressed and stuck out at an unnatural angle from the skull. Several large vessels could be seen over the surface of the pendulous posterior portion.

Fig. 1.—The patient in November, 1930, before operation.

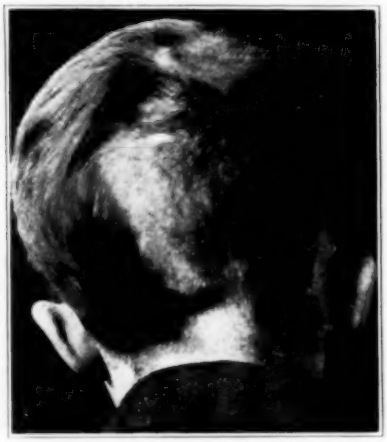

Fig. 2.—The patient after operation.

Pulsations synchronous with the beat of the heart were detected at the posterior border of the mass. A bruit was not heard, nor was there fluctuation. At this time biopsy was suggested, but was refused by the parents.

Second Hospitalization.—In November, 1930, when the patient was 17, he returned to the clinic, much inconvenienced and very anxious about the mass hanging down behind his ear. A few months before his return he had been in an automobile accident in which lacerations over the tumor had been sustained. The tumor was

larger and firmer than at the time of his previous visit. The pendulous mass now measured about 15 cm. transversely and was about 10 cm. thick. There was a small area of ulceration on the lateral aspect. The pulsations synchronous with the heart beat were still present, although they were not so marked. The right ear had suffered still further displacement downward and outward, and altogether he presented a sorry spectacle. The patient had always been regarded as normal except that his head was larger than usual. He had walked at 1½ years of age and had talked at 2 years. He expected to graduate from high school in June, 1931.

On roentgenologic examination, there was found an area of destruction of bone, 4 by 6 cm. in diameter, in the right occipitoparietal region, involving the right temporoparietal and the right occipitoparietal sutures. There was hydrocephalus of the right side and deformity of the skull. A large, soft mass of tissue could be seen. Vision in the right eye was 6/20 and in the left 6/60. The optic disks were

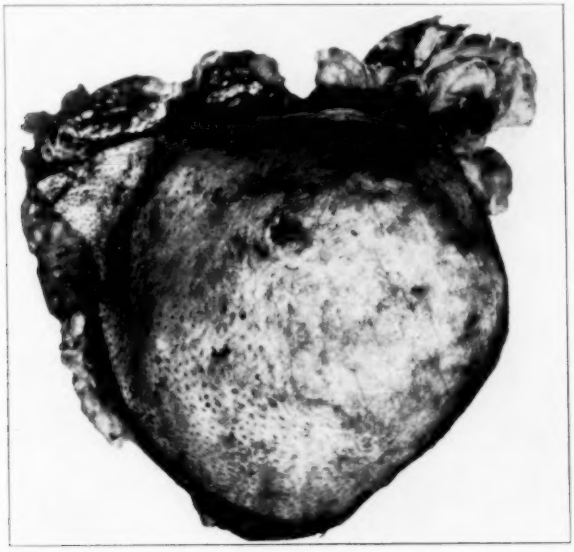

Fig. 3.—The mass removed at operation; the area of ulceration is evident.

structurally full; there was some marginal blurring, and there were dilated retinal veins, especially inferiorly in the left eye. Soft tumors involved the right lower lid and widened the palpebral fissure. There was no evidence of intra-orbital involvement. Other examinations gave negative results.

In differential diagnosis, sebaceous cyst, lipoma, meningocele, hemangioma (partially thrombosed) and neurofibroma were considered, but diagnosis finally lay between hemangioma that had become partially thrombosed and neurofibroma. It was decided to remove the mass and thus relieve the patient of the main cause of disfigurement and avoid ulceration and necrosis of the superficial part of the mass.

The mass was removed, with the skin over it. It was found to communicate with many large vessels, most of which were thrombosed. There were many nodules scattered throughout the tumor, which resembled neurofibromas. The tumor did not communicate with the cranial contents; these were covered by dura mater. Before the operation was completed, the ear was stitched in a more natural position.

The patient was to return for a second operation on the part of the tumor that remained in the right temporal region and that extended into the right orbit.

The tumor removed was a malignant neurofibroma. Most of the excised mass presented the typical appearance of a neurofibroma, but in the softer parts there were cells of variable sizes and shapes, mitotic figures and some giant tumor cells (figs. 3, 4 and 5).

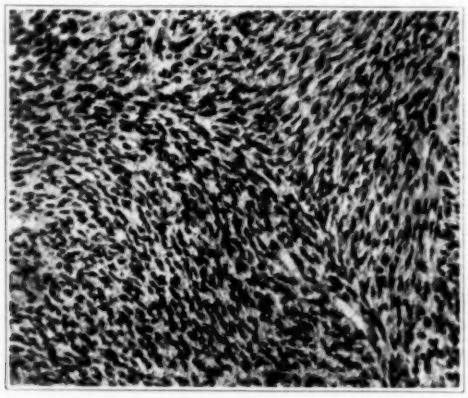

Fig. 4.—Section from the firmer portion of the tumor; the structure is fairly typical of neurofibroma, with interlacing of cells, and so forth; × 125.

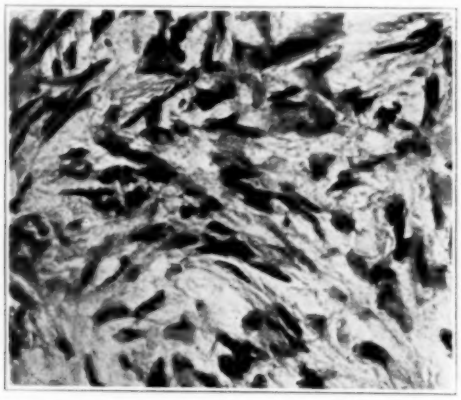

Fig. 5.—Section from the softer portion of the tumor; the irregular shape of tumor cells, mitotic figures, and so forth, are seen; × 350.

COMMENT

Before operation it was almost impossible for us to decide that we were not dealing with a tumor of a blood vessel and to determine whether it communicated with the intracranial blood sinuses. The pulsations that were observed probably were due, in part, to pulsations transmitted from the brain through the dura, and in part from the large, open vessels that communicated with the posterior portion of the tumor. The history and the feel of the tumor favored the diagnosis of neurofibroma of the scalp.

In 1906, Helmholz and Cushing [1] reviewed the literature on these tumors and reported a case. They gave a detailed account of the cases that had been described up to that time, and showed that in almost all of them the condition was congenital. They mentioned some of the many names under which this type of neoplasm has been discussed. Reports of about sixteen cases of fibrosarcoma of the scalp are to be found in the literature, some of which resemble very closely the case of neurofibroma seen by us. The first case of sarcoma of the scalp was reported by Conner [2] in 1888. The history dated back fifteen years, to childhood, and the mass involved most of one side of the head. Recently, Craig [3] reported a case of neurofibroma of the occipital nerve which resembled in many ways the case presented here. It would appear, then, that certain neurofibromas may become malignant.

The prognosis in this case is indeterminate on account of the malignant changes found in the tissue removed. However, the remaining part of the tumor should be removed to prevent optosis. In view of the fact that this neoplasm is of such slow growth, further operations may be advised should there be recurrences.

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1. Helmholz, H. F., and Cushing, Harvey: Elephantiasis Nervorum of the Scalp; a Manifestation of von Recklinghausen's Disease, Am. J. M. Sc. **132**:355 (Sept.) 1906.

2. Conner, P. S.: Case of Sarcoma of the Scalp, Ann. Surg. **8**:110, 1888.

3. Craig, W. McK.: Angioneurofibroma of the Occipital Nerve, Am. J. Surg. **2**:593 (June) 1927.

News and Comment

STUDY OF MENTAL HOSPITALS BY THE AMERICAN MEDICAL ASSOCIATION

The Council on Medical Education and Hospitals has extended its study of general hospitals, which has been carried on for many years, to include hospitals for mental illness. This work is being conducted with the advice and assistance of a committee on mental health, appointed by the Board of Trustees of the American Medical Association, composed of Dr. H. Douglas Singer, Chicago, chairman; Dr. F. G. Ebaugh, Denver; Dr. E. J. Emerick, Columbus, Ohio; Dr. J. Allen Jackson, Danville, Pa., and Dr. W. L. Treadway, Washington, D. C. The first step has been a fact-finding survey by questionnaire of all listed private and governmental institutions for the treatment of patients with mental illness, which was sent to 561 institutions in October, 1931. Personal visits have also been made to many of these institutions by Dr. John M. Grimes, who was specially appointed for this work, and other members of the Council's staff of visitors, primarily to establish personal relations between the Council and the officers in charge of the institutions, and secondarily to afford an opportunity for a bird's eye view of the facilities provided in each institution for hospital service. The visitors are all physicians experienced in the inspection of hospitals, particularly as regards their suitability for the training of interns and residents. A report of the work done and the amount of material collected was presented by Dr. Grimes at the Annual Congress on Medical Education, Medical Licensure and Hospitals in Chicago on Feb. 16, 1932, and was printed in *The Journal of the American Medical Association* (**98**:839 [March 5] 1932). This report showed that returns at that time were already 75 per cent complete.

On the recommendation of the advisory committee, and with the assent of the Council, the Board of Trustees of the American Medical Association has appropriated a sum of money to pay for the services of a competent psychiatrist to study the returns to the questionnaire and to summarize the results of previous surveys that have been made by various agencies, so that they may be available for the use of the Council in planning the next steps to be taken. The Council has requested the committee to nominate a psychiatrist to fill this position. Provision has also been made for the closest possible cooperation between the committee and the Council.

The general purpose that led to the institution of the study, of which the questionnaire and visits thus far made represent only a preliminary step, is to afford a national backing for hospital managers in their efforts to improve the standards of service in these hospitals and to render them available for training physicians in psychiatry, a field that is being increasingly recognized as an important one in the general practice of medicine.

CORRECTION

In the discussion of Dr. Stanley Cobb's paper "Causes of Epilepsy" in the May issue (**27**:1245, 1932), in the second paragraph on page 1263, the words "from 2 to 3 liters of fluid is produced" should read "from 500 to 1,000 cc. of fluid is produced."

Abstracts from Current Literature

THE QUESTION OF NARCOLEPSY. E. REDLICH, Ztschr. f. d. ges. Neurol. u. Psychiat. **136**:128, 1931.

This publication, given to the medical world after the death of Redlich, contains his later views on the subject of narcolepsy. In it he has gathered all the available material and has analyzed it with reference to its outstanding problems. The question arises as to why the disease, which was formerly so rare, is now seen so frequently. Wilson believes that it has become more common in recent years. Others report that it often follows encephalitis and look on it as a post-encephalitic phenomenon. Redlich says that he saw his first case in 1914, long before encephalitis appeared, and that the latter cannot be looked on as an etiologic factor in the production of narcolepsy. He thinks that more cases of narcolepsy are reported now because, through the numerous reports which followed his, physicians are much better acquainted with the disease.

Men are much oftener affected than women. Of 100 patients, 79 were men and 21 were women. Of these same 100 cases, four occurred before 10 years of age, 50 between 10 and 20, 23 between 20 and 30, 18 between 30 and 40 and 3 after 40. That is to say, 54 per cent of the cases occurred before 20.

In several cases a familial hereditary tendency has been noted. Often, however, in the other members of the family the condition was not true narcolepsy but an increased need for sleep. Redlich could find only eight patients with a constitutional hereditary tendency, and among the families of these patients one was often not dealing with true narcolepsy. A history of alcoholism was often obtained in the forbears. Psychoses have been reported among the families of narcoleptic persons; there were psychopathic tendencies in the family of Curschmann's patient, among whom two committed suicide, and in other cases the patient had forbears with depressions and paranoid psychoses, some of whom died in institutions. Multiple sclerosis was reported in the brother of one patient. Trauma of the head has been reported in a large number of cases. This has been slight or has occurred some time before the onset of the narcolepsy. Severe trauma of the head, followed shortly by narcolepsy, has been reported by Redlich, Goldflam and Haluel.

Objection has been raised to the term narcolepsy because it implies paralysis, and Singer has suggested the term hypnolepsy. This is open to objection because it refers only to a limited aspect of the disease, the attacks of sleep. In order to avoid the controversy of terminology it has been suggested that Gélineau's syndrome be the term used. This, too, is inaccurate because cases were reported before Gélineau by Fischer and Westphal. To be strictly accurate one should speak of the Westphal-Gélineau syndrome. Redlich prefers the term narcolepsy.

All the attacks of sleep in narcolepsy have the characteristics of true sleep. They are preceded by an irresistible desire for sleep; some attacks may be averted by brisk walking in the fresh air, by powerful sensory stimuli or by dashing cold water over the face. Often the attacks are preceded by protracted yawning, and Wilson reports a marked loss of affective tonus before attacks. Patients often complain of generalized paresthesia before an attack. Often, too, motor phenomena precede, such as twitching of the eyelids, movements of the hands or of the jaw and slight movements of the trunk. In a case of Fischer the sleep was preceded by hypnagogic hallucinations. During the attack the patient has all the characteristics of a person in true sleep: the face is slightly congested, the attitude is relaxed, and inspirations are somewhat slowed and increased in depth. The sleep attacks usually occur when the patient is quiet and alone, in his room

or in a garden, a street car or a railway coach, and particularly when he is in bed. Often narcoleptic persons fall asleep in company, especially when listening to speeches. They fall asleep walking or standing and wake up when they bump something or stumble. Singer and also Fischer reported that patients fell asleep during coitus. Dreams occur during the sleep and can often be recalled. The duration of the sleep is usually short; often it is only a few minutes, sometimes it is longer, but rarely is it an hour or more. Narcoleptic persons can be aroused easily by light shaking. On waking they look about in astonishment, rub their eyes, become conscious again immediately and continue their work. The frequency of the attacks varies; Gélineau's case of 200 attacks a day is extreme. From 5 to 6 attacks a day is about the average, the attacks usually occurring from 9 to 11 a. m. or after 5 p. m. Many narcoleptic patients feel better in winter and worse in summer. Sometimes attacks can be induced by hyperventilation.

The night sleep of narcoleptic persons is not good. There are often much tossing and turning, pavor nocturnus and wandering about at night, reminiscent of somnambulism. Sometimes these phenomena precede the narcolepsy; often they begin in early childhood. Talking during sleep occurs frequently among these patients.

The inhibition of the affect is one of the most interesting features of narcolepsy. Laughing even among normal persons is attended by a certain loss of tonus. In narcoleptic patients laughing is the most effective stimulus for bringing on attacks, but anger, fear and worry are also effective. Tickling does not produce them. The recalling of an operation or of some incident attended by danger is most effective in producing attacks characterized by drooping of the lower jaw, lowering of the head on the chest, buckling of the legs and trunk and flaccidity of the extremities. The attacks last for from two to three minutes; afterward the patient is as active as ever. In severe cases there is a fleeting loss of tendon reflexes (Wilson). Consciousness is not interfered with during the attacks, and narcoleptic persons are fully aware of what goes on around them. Knowing the effects of laughter on them, these patients try as much as possible to suppress it, and even go so far as to avoid company in order to prevent attacks. There is an actual loss of tonus during the attacks (Redlich). The fleeting loss of reflexes seen in these cases speaks for a loss of tonus. The attack is not true catalepsy. Redlich warns against such terms as lachschlag, geloplegia, and the like, because the attacks are brought on by emotions other than laughter. He does, however, offer the concept of loss of tonus in these attacks with certain reservations, because there have been reported some phenomena that cannot be explained on this basis. Tremor and a drawing over of the face have been seen in a few cases (Westphal, Loewenthal). Peculiar movements of the head (Noak), spasm of the tongue (Goldflam) and generalized spasms (Fischer) with intact consciousness have been reported. These phenomena occur usually in the beginning of attacks before the onset of loss of tonus and may be looked on either as a sort of tonic spasm or as clonic movements. Tremor of the lips and lids and deviation of the jaw have been reported.

The sleep attacks and the affect tonus loss are closely related. There are patients in whom affect changes bring on sleep, the latter coming on spontaneously only later. In the great majority of patients both types of attacks are present. This was true in 72 per cent of 100 cases. In 12 cases there was no loss of affect tonus, and there were a few cases of this sort that were definitely postencephalitic.

As to the neurologic status of narcoleptic patients, slight differences in the tendon reflexes have been reported. In a case of Rosenthal's there was a Babinski and an Oppenheim phenomenon, and in a case of Goldflam there was deviation of the tongue to the left. Brook and also Zehrer have reported a sympathetic ophthalmoplegia. Wilson and Kennedy reported diplopia in their cases. One of Redlich's patients had ticlike spasms of the platysma and suffered

from migraine. Nystagmus has been reported in a few cases, as have also vertigo and enuresis. Lumbar puncture in the cases reported has always given negative results.

From the vegetative and endocrine standpoint there are interesting observations. The habitus varies; in some cases there is a typical picture of a lymphatic constitution. Redlich pointed out the relation of the pituitary gland to narcolepsy, since cases of pituitary tumor are so often accompanied by drowsiness. He has seen a patient with pituitary tumor who had attacks of narcolepsy. An extremely small sella has been reported in several instances of narcolepsy; Redlich has seen it in 4 cases. Dercum found an unusually large sella in his case; the patient had acromegalic features. An enlarged thyroid is sometimes present. In a few instances hypothyroid features have been recorded. The genitals are often poorly developed, and secondary sex characters are poorly defined. The basal metabolism has been reported now in a number of cases, and Redlich says that it is almost always, if not always, decreased.

Eosinophilia (from 4 to 7 per cent) has been found in a few cases. Not infrequently, as Redlich pointed out, there is lymphocytosis of from 40 to 50 per cent, sometimes less. Cases have been reported in which the lymphocyte count was normal. There is lymphocytosis in most cases of narcolepsy according to Redlich, particularly in young patients. It is found often in postencephalitic cases. Its significance is not clear.

There is often fatty diathesis in cases of narcolepsy, setting in soon after the disease appears. It appears to be associated with decreased metabolism. The temperature is usually normal, but Bogaert reported a case in which the temperature dropped during attacks of sleep. Polyuria and polydipsia have been seen in some cases. The blood calcium is normal, and so, too, is the blood sugar.

As to prognosis, the disease is chronic, with a tendency to amelioration during the course of the years and even a remission in some cases. All sorts of therapy have been tried with little effect. Thyroid extract has given the best results.

ALPERS, Philadelphia.

FRACTURE OF THE SKULL IN CHILDREN. JAY IRELAND, Arch. Surg. **24**:23 (Jan.) 1932.

Eighty cases of proved fracture of the skull in patients under 12 years admitted to the Children's Memorial Hospital from 1913 to 1930, inclusive, are reported; fifty-four were boys and twenty-six, girls. The youngest was 46 days old; the oldest, 11 years; the average age was 4 years. From the ages of 9 to 10 and from 10 to 11 years, the smallest number of children were injured; there were two in each age group. The greatest number, thirteen, occurred in the group from 3 to 4 years of age. At this age, gait is unsteady and falls are frequent; consequently fracture of the skull is more common. Falls produced 62.5 per cent of the injuries, and automobiles 25 per cent. Many of the falls were from apparatus on play grounds. Among other causes were blows, street car and bicycle accidents, crushing injuries and gunshots.

An analysis of the site and extent of the fracture is given. Sixty-three patients (78.7 per cent) had fractures of the vault alone; eight (10 per cent) of the base alone, and eight (10 per cent) of both vault and base; the location in one (1.2 per cent) was not stated. Only two showed separation of the sutures. The percentage of fractures of the basal skull in children seems lower than in adults. The parietal bone was most frequently fractured. The frontal bone came next in frequency. Eight patients had depressed fractures. Three patients had definite elevation of the fragments, which is rarely described in the literature.

The longest fracture completely encircled the vault and measured 36 cm. This fracture was still plainly visible by x-rays six hundred and ten days after the injury. Eleven patients were reexamined roentgenographically. The shortest period between the first and second examination was two hundred and eighty-two days; the longest six years and twelve days, and the average six hundred and

forty-seven days. In every patient except the one mentioned, the fracture had completely closed at the second examination, without evidence of thickening or callus.

The incidence of signs and symptoms is analyzed in detail; thirty-two (40 per cent) patients were rendered immediately unconscious by the accident; twelve (15 per cent) were unconscious on admission; eighteen (22.5 per cent) regained consciousness between the time of the injury and admission; sixty-eight (85 per cent) showed visible laceration, hematoma or contusion of the head; seventeen (21.2 per cent) had headache; nine (11.2 per cent) had dizziness; fifty-one (63.7 per cent) vomited, and eighteen (22.5 per cent) had bloody vomitus. From this, vomiting appears to be an important symptom of fractured skull in children.

In sixty-six the pupils were equal, in eleven unequal; in two fatal cases they were dilated and fixed. The fundi were examined in six patients; in three they were normal; one had a hazy disk; one a definite papilledema; one a retinal hemorrhage; two patients had paralysis of the extrinsic eye muscles; three had nystagmus and one diplopia; nineteen had bleeding from the nose, which was most common in fracture of the frontal bone; eleven patients had bleeding from the ears, one with a fracture of the base, seven of the vault, two of both base and vault, and in one case the site was not stated, and fourteen patients bled from the mouth. The observations indicate that normal reflexes frequently accompany fractured skull in children.

The author believes that "no definite prognostic conclusion could be drawn from the blood pressure of these patients." The highest recorded pulse rate was 180 and the lowest 50; the general average was 106. From a consideration of the cases reported, the author suggests that a high pulse rate (over 150) is not a bad sign in fractured skull in children. The highest respiratory rate recorded was 60, the lowest 15; the general average was 26.5. The author states that "no definite prognostic conclusions can be drawn from the temperature in these cases, unless it is that cases marked by high temperatures (over 102 F., taken rectally) are not usually fatal in children, as they often are in adults."

The author believes that lumbar puncture should not be done for diagnosis if this can be made by physical examination, roentgen examination or other means. No case was reported with a diagnosis of epidural hemorrhage. "Epidural hemorrhage is less frequent in children than in adults, because the dura is more adherent to the skull in children, while in adults it is loosely adherent and allows a clot to form readily between it and the skull." One patient had a fracture through the frontal sinus; infection of the brain did not follow.

The author gives reasons for considering fractures of the skull in children as having a better prognosis than in adults. The mortality in the eighty cases presented was 2.5 per cent. The author compares this low figure with other statistics. He remarks that: "The injury to the brain is much more important than the fracture of the skull."

Ireland reviews briefly the question of administering hypertonic solutions and doing lumbar puncture in the treatment in conditions involving the head. He believes that: "Therapeutic lumbar puncture should not be done as a matter of routine in cases of fracture of the skull in children." Again, "With signs of increasing pressure, it may be carefully done, the fluid being drained off very slowly and the drainage being discontinued if any untoward symptoms develop." If relief is not obtained, surgical decompression may be done.

Ireland discusses the type of operation with references to the literature, and adds: "All large bony fragments, except those definitely contaminated, should be replaced, even if completely loosened from all tissue at the time of operation." He believes that, in children, small depressions only slightly below the surface of the skull and those involving the nasal sinuses or superior sagittal sinus are often best treated without operation if they produce no symptoms. All the patients in the cases reported were kept in bed for four weeks as a routine. Ice packs were placed on the head on entrance to the hospital. Sedatives were used for pain and shock. Ether had to be given to one patient for convulsions, and stimulations

were given to another. Tetanus antitoxin was given when indicated. In eighteen (22.5 per cent) no medication was given.

Follow-up studies for various periods, ranging from one day to six years and eleven days after injury, showed that sixty-nine patients had completely recovered: nine (11.2 per cent) had serious sequelae when last examined, as follows: epilepsy, one; paralysis of the left arm and enlargement of the left pupil, one; weakness of the left leg, one; left leg weaker and smaller than the right, one; staggering gait, one; paralysis of the right side of the face and troublesome pulsation in the right ear, one, and headache, three. Some statistics on the sequelae to trauma of the head are added. Ireland summarizes as follows: "1. Eighty cases of proved fracture of the skull in children, with 2 deaths (2.5 per cent) are discussed. 2. In all cases, except 7, the treatment was conservative, without decompression. 3. Seven patients with depressed fractures and 3 patients with elevated fractures were treated without operation. All recovered. 4. Lumbar puncture should not be done for diagnosis when diagnosis can readily be made otherwise. 5. Children suffering from fracture of the skull should, in most cases, be treated conservatively."

The large series of cases is most instructive and again raises the question of the value and relative danger of lumbar puncture in injuries of the head in children. The average length of time for which the patients have been followed is three hundred and ninety-six days, which perhaps is still too short a period on which to base definite conclusions as regards ultimate sequelae.

ELVIDGE, Montreal.

A CONTRIBUTION TO THE PROBLEM OF HEREDITY AMONG SCHIZOPHRENICS. M. BLEULER, J. Nerv. & Ment. Dis. **74**:393 (Oct.) 1931.

The author undertook a study of 100 cases of schizophrenia from the Bloomingdale Hospital, in which the diagnoses were undisputed, as well as the study of 2,634 relatives of the 100 patients. Cases were eliminated in which information about the family was obtained with great difficulty. The family histories of other physicians were utilized, though the author recognizes the inadequate investigation of apparently normal relatives which such material entails. The grandparents, uncles and aunts were not all included in the investigation, but it was possible in the great majority of instances to obtain information about them. However, the study of parents, siblings and children of the patients was complete. In the group selected there were 69 men and 31 women with an average age of 34 and 42 years, respectively. Socially, the majority of the cases belonged to the intellectual or well-to-do classes. E. Bleuler's conception of schizophrenia was adopted, and in some of the doubtful cases he made the diagnosis himself. Of the 100 patients only 30 showed clear signs of dementia. Among the 351 siblings of the 100 patients there were nine cases of schizophrenia and three cases of suspected schizophrenia, a somewhat higher incidence than that found by Ruedin. The author's figures agree with those of Luxemburger, who found an expectation of illness of 5.1 per cent in siblings of schizophrenic parents. This agreement is interesting in view of the racial and social differences in the case material of the two authors. The majority of the author's patients have above average intelligence and higher education, and in his cases the low percentage of 0.28 offers some contrast to 0.6 per cent of feebleminded in Schulz' cases and 4.9 per cent of Kattentidt's. Among the siblings of Bleuler's 100 cases there were 39 psychopathic and markedly eccentric persons, constituting 13.6 per cent of all siblings over 10 years of age. There was a percentage of alcoholism of 1.6, comparable to Weinberg's 2 per cent.

Among the 200 parents there were 4 persons with schizophrenia, 3 with possible or probable schizophrenia, 5 with senile and postapoplectic psychoses, 5 with inadequately known states of depression at an advanced age, and 1 with delirium tremens. In other words, a total of 18 with psychoses, or 9 per cent. Hoffman found that 8.4 per cent of parents of schizophrenic children had psychoses, and

Weinberg found that 14.8 per cent of the parents had this condition. Hence the frequency of schizophrenia among the parents in Bleuler's cases is about the same as was found in Germany. Clear manic-depressive psychoses were not found among the parents; but a careful investigation showed that 23 per cent of them were psychopathic and markedly eccentric—a higher incidence than among the siblings. One and five-tenths per cent of the parents ended their lives by suicide. From 15 of the 200 parents no data of their siblings could be obtained. Among the 949 members of the generation of the patient's parents, only six pairs of twins were known, a lower incidence than normal. Among these siblings of parents there is a roughly empiric 1.34 per cent of schizophrenia and strongly suspected schizophrenia, and the expectation of illness from schizophrenia for the uncles and aunts of schizophrenic patients is 1.64 per cent. Among the collateral lines there are 82 psychopathic and markedly eccentric persons, or 12.3 per cent of those over 10 years of age.

Forty of the 400 grandparents remained wholly unknown, but in the remaining 360 there were 15 with psychoses, eight of which were senile psychoses and only 1 was certainly schizophrenic. Of the 128 nieces and nephews, only 52 were over 18 years of age. One case of psychosis existed in this group, and that patient was severely schizophrenic. Among 495 cousins of the patients, 10 psychoses occurred, including one case of positive and three cases of very probable schizophrenia. Of the 30 children of the patients only 10 had passed the twentieth year and were not considered suitable for statistical study.

The question as to whether heredity plays the same rôle in all forms of schizophrenia was a difficult one for the author to answer. He separates his patients into two groups, those without a hereditary strain and those with one. Patients with a strain were those who had schizophrenic patients among the most immediate relatives. Pronounced eccentrics of schizoid type are also included, but not the organic psychoses or the milder eccentrics. He finds 76 of the cases showing an evident psychotic strain, a transition group of 16 showing some eccentricities which were not marked among the relatives and a group of 8 patients showing no psychotic strain or even eccentricity. These figures agree closely with those of Wolfsohn, 90 per cent, and are higher than those of Barrett, 78 per cent. The patients without a psychotic strain showed a remarkable feature, namely, more severe catatonic dementia than the cases with hereditary taint. Six of the 8 patients showed severe dementia, but nevertheless there was possible a kind of affective rapport as well as a capacity to conform to simple regulations. Their attitude seemed less tense and peculiar than many persons with schizophrenic dementia. Five of the 8 were dysplasic and obese. A study of the prepsychotic personality in these 8 patients showed a certain uniformity. They lacked schizoid idiosyncrasies and were good natured, but were indolent and lacked adequate initiative. In these 8 cases, 7 patients were perfectly normal before the psychosis; only 1 showed schizoid eccentricities. On the other hand, among the 92 affected patients, 45 were strongly eccentric before the psychoses. Hence the author's findings contradict the old view that dementia is the essential factor in schizophrenia, and seem to agree with those of the old Zurich school that a single severe case of schizophrenia does not indicate such a bad prognosis for descendants as several light cases of schizophrenia or schizoid psychopathy, though Kohn has maintained that the schizoid state is directly inherited and Hoffman found almost always a schizoid partner among the patients of schizophrenic patients. In the author's material there were several clear instances of schizoid and schizophrenic persons whose two parents were not schizoid but who were classic examples of syntonic personalities.

The author concludes that the appearance of schizophrenia in families has not been proved to be determined by heredity. The data available on twins, he considers, show that heredity cannot be the only cause. If anything is inherited in schizophrenia, it is least of all the schizophrenic dementia. The author supplements his observation with histories of patients, siblings, parents, grandparents and other relatives in considerable detail to illustrate more accurately the material and data from which his conclusions are drawn.

HART, Greenwich, Conn.

THE BRAIN IN A CYCLOPEAN GOAT. WALTER STUPKA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **33**:315, 1931.

The two most striking abnormalities were found in the visual and in the olfactory apparatus. It was difficult to find the peripheral portion of the optic nerve, but the central part was evident. The extra-ocular muscles were well innervated and unusually well developed. The solitary eyeball had a bilateral symmetrical muscle and nerve supply; i. e., its muscle and nerve supply was the same as if there had been a pair of eyes. The lens was unusually small and enclosed in a thick connective tissue capsule. Across the lens there were small muscle bundles running in either direction, attached to unusually well developed eyelids. The ciliary body, vitreous, choroid and sclera were normal. There was total absence of the peripheral olfactory apparatus.

Macroscopically, the brain appeared normal up to the superior quadrigeminal bodies, and it was not until the thalamus was reached that abnormalities came into evidence. Genuine malformation really began to be noted in the midbrain. The secondary forebrain was poorly developed, and no cerebral hemispheres were seen. These two abnormalities led to an abnormal relation of the basal ganglia to the brain stem; this abnormal relationship resembled closely that observed during early fetal development.

In spite of the absence of the fila olfactoria, there was present orally and ventrally a portion of the brain that apparently belonged to the olfactory apparatus. This structure was connected by a bridge to the remaining portion of the forebrain, was indented and probably represented the olfactory lobe on either side of which a bundle of nerve fibers was seen to run caudad and ventrad. It was apparent that these fibers were attached to the fibers of the internal capsule, where they lost their identity so that they could not be traced any farther. Ventrally, there were also observed two column-like formations that probably represented the septum pellucidum. The cornu ammonis and hippocampal regions were well developed and seemed to be in relation with whatever olfactory apparatus existed.

Horizontal bundles of fibers ran transversely between the thalami where one would ordinarily expect to find the fornix. It is unlikely, however, that these fibers were the fornix because they were exhausted as one attempted to trace them orad, although some of them, at least those running in a caudal direction, seemed to terminate in the corpus mammillare. There is no question but that there existed a fasciculus thalamomammillaris, which showed some indefinite connections with the tegmentum mesencephali and the ganglion interpedunculare. The taenia thalami was strikingly well developed and could be traced to a massive ganglion habenulare, which was connected by a commissure with the opposite side. Corresponding to the well developed ganglion habenulare there was also present a massive fasciculus retroflexus, which could be followed into the ganglion interpedunculare. The fact that the corpora quadrigemina were exceptionally well developed and the thalamus was less so led to a partial roofing of the latter by the quadrigeminal plate, so that the ventral portion of the thalamus seemed to begin more caudally than is normal. The pedunculus corpori mammillaris was situated bilaterally and symmetrically, and was very well developed; part of it could be traced to the region of the ganglion interpedunculare and the other part dorsally. Gudden's tegmental ganglia were also situated bilaterally and symmetrically and were unusually well developed.

The nuclei of the extra-ocular muscles were situated normally and were well developed. The cochlear and vestibular apparatus, as well as the remaining cranial nerves, showed no abnormalities.

There is no question as to the existence of a lemniscus and of the nuclei of Goll and Burdach. The lemniscus, however, though appearing normal, was perhaps situated in the medulla more dorsally than is usual, and its termination in the thalamus could not be definitely determined. There was a total absence of pyramidal tracts, as a result of which the two well developed olives appeared on the surface of the medulla as if they were not separated from each other by an interolivary layer. The absence of pyramidal tracts in the pons, as well as the absence of

cerebral peduncles, led to a peculiar appearance of the substantia nigra which could be recognized only on its ventral surface.

Owing to the peculiar arrangement of the thalamus, there was great difficulty in making out the internal capsule.

A study of the fiber tracts to and from the basal ganglia revealed the following connections: (1) centripetal fibers from the thalamus to the cortex; (2) crossed and uncrossed connections between the thalamus and the striopallidum; (3) crossed corticothalamic fibers, and (4) fibers that could be readily traced into the pons.

The structure of the cerebrum was very simple; only four convolutions could be seen, although they could not be clearly distinguished; this was due to a defective differentiation of the various layers. A corona radiata, an indication of a corpus callosum and an unusually well developed psalterium could readily be made out. Caudad, i. e., in the region of the psalterium, there was a uniform median ventricle that extended laterad and ventrad to the region of the cornu ammonis. Orally, an unpaired cavity alone could be seen.

Owing to the unusually well developed olives, the central tegmental tract was correspondingly well developed, although its site of origin could not be determined. There existed a definite olivocerebellar connection, a restiform body and a large median cerebellar lobe. The cerebellar nuclei showed no abnormalities, although the lateral cerebellar lobes seemed somewhat underdeveloped. No abnormalities were noted in the brachium conjunctivae and in the nucleus ruber system. In contrast to the poorly developed cerebral hemispheres, the pons was very large, and although its transverse fibers could be made out only with difficulty, there is no question but that such fibers existed and that the perpendicular fibers to the tegmentum pontis and pontile nuclei were strikingly well developed. There was a reduction in the pontocerebellar fibers and almost complete absence of the pontocortical fibers.

The paper is concluded with a discussion of the genesis of this type of cerebral malformation; it should be read in the original by those interested in the subject.

KESCHNER, New York.

CHIASMAL SYMPTOMS IN INTRACRANIAL TUMORS. LOYAL DAVIS, Arch. Ophth. **6**:181 (Aug.) 1931.

The author begins this article with a discussion of rather important points in the technic of cranial surgery. Though they may seem trifling, they contribute to the entire picture of good neurologic surgery. He follows this discussion with a plea for microscopic verification and classification of the tumors exposed and a correlation of the pathologic picture with the clinical course before and after operation. He then proceeds to the symptomatology of the intracranial tumors under consideration in his article: (1) homolateral optic atrophy, contralateral papilledema and anosmia; (2) bitemporal heminaopia and optic atrophy in a middle-aged person with a normal sella turcica; (3) unilateral exophthalmos and optic atrophy; (4) indefinite defects in the fields, optic atrophy and rapid progressive loss of visual acuity; (5) definite defects in the fields, papilledema and rapid progressive loss of visual acuity.

Group 1, a syndrome of homolateral optic atrophy, is known as the "Foster Kennedy syndrome," because Kennedy called attention to it in 1911. It is illustrated by the history, physical examination and operation in a case. It is of interest that in this patient the bilateral anosmia was uncovered only by examination. The patient was unaware of it.

Group 2, the classic "Cushing chiasmal syndrome" was illustrated in the same way. The first case was one of suprasellar meningioma, the second, one of craniopharyngioma. In these cases the calcification in the latter is emphasized, while in the fourth case of this group the author calls attention to injury of the hypothalamus in the surgical removal of these tumors.

The third and fourth groups of cases (craniopharyngioma, glioma and meningioma) were illustrated by three different patients, and outlined in great detail.

The first case was one of meningioma, the second glioma and the third a partly removed meningioma. In the case of the glioma, the operation was unfavorable in that the tumor extended back into the third ventricle. The patient was a child aged 5. The case of meningioma was of great interest in that the exophthalmos receded very shortly after the operation, and the patient had fairly satisfactory vision in one eye. In the third case of a partially removed meningioma, the patient had three subsequent operations: the first was a simple decompression; at the second, a partial electrosurgical cautery was done on the right side; at the third, five days later, a flap was reelevated on the left side, and a large soft tumor mass was removed in part. Five days later, another attempt was made to remove the remainder of this enormous tumor. It was evident, however, that the condition was no longer operable. The patient died three months later. In this case the visual fields were so contracted that no localizing assistance was possible. This was exactly the opposite in the cases that came within the second group of the author's classification.

The fifth group was illustrated by two different cases. The first was that of a child, aged 7, totally blind, with 5 diopters of papilledema on the right and 3 diopters on the left, and with a normal sella turcica. An operation was performed, and subsequent microscopic study revealed a hyperplastic inflammatory arachnoiditis. Visual acuity after complete recovery was 20/50 in both eyes. The fields of vision showed an irregular concentric contraction. There was considerable atrophy of the optic nerve after full recovery. The second case of this type was one of meningioma, which at autopsy was found attached to the falx cerebri between the occipital lobes.

The author's comment in completing the paper was a consideration of 252 intracranial tumors, in 191 of which the diagnosis had been verified by microscopic section. Of this group, 14 fall within the classification of chiasmal lesions. The operative mortality rate was 14.2 per cent. Nine of the 10 patients still living are at this time socially and economically independent. SPAETH, Philadelphia.

COMPARATIVE ANATOMY OF THE VESTIBULAR NERVE. KANICHI ISHIHARA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **33**:233, 1931.

The structure of the vestibular nerve seems to be remarkably uniform in all animals. The nerve can be recognized in a large number of animals as an isolated lateral fasciculus running in a dorsal direction through the most lateral portion of the restiform body. In most animals the fasciculus bends at the dorsal margin of the restiform body mesially to reach partly the area triangularis and partly the area angularis. In carnivora and in rodentia the bundle can be traced upward, where it is seen to run with the restiform body toward the cerebellum. In practically all animals the main portion of the nerve lies mesially to the restiform body. Its course depends on the relation of the spinal root of the trigeminus to the restiform body. Depending on the position of these two structures, the vestibularis runs from its ventrolateral position in the dorsomesial direction, it bends (in the hedgehog and in Ungulata) after a short vertical course in the horizontal direction, or it assumes an S-shaped course (in Delphinia).

The nuclei commonly situated in the ventral portion of this isolated fasciculus can generally be recognized as components of the cochlearis. It is only in some animals that it seems as if some fragments from the substantia gelatinosa trigemini are deposited in this portion of the vestibularis.

As the vestibularis reaches the region of the area fasciculata it divides, in most animals, into an ascending and a descending branch. The descending branch is always situated in the ventromesial portion of the area fasciculata and consists of a bundle formation which is not as dense as that of the area fasciculata. It is not always easy to trace the ascending branch of the vestibularis, because of its close connection with the nucleocerebellar and fastigiobulbar systems, although in a few cases terminal vestibular fibers may be found in the area angularis and in Deiters' nucleus, especially in its most lateral and oral portion.

Dorsally, the area angularis is adjacent to the area fasciculata; its dorsal boundary is the brachium conjunctivum; ventrally, it is situated near the area fasciculata; laterally, near the restiform body, and mesially, near the ventricle.

In addition to ganglion cells, the vestibularis consists of three bundles of transverse fibers running dorsally, ventrad from the brachium conjunctivum, and occasionally ventrolaterad to it. These bundles are coarse systems of fibers corresponding to those of the area fasciculata; they are apparently the fibers that bend from their straight vertical course in a sagittal direction in order to reach ventrally the nucleocerebellar and fastigiobulbar systems. In the Pteropus, and also perhaps in other species, the area fasciculata contains fibers that are related to the trigeminus.

A second system of fibers from the vestibularis may also be seen bending in a sagittal direction in order to form a more diffuse fiber system; these fibers, however, do not run caudad, but orad, and reach the most oral portion of the angularis nucleus, or they surround mesially the brachium conjunctivum in order to join the fasciculus uncinatus with which they enter the cerebellum. It is also possible that one may be dealing here with secondary tracts from the nucleus angularis. Finally, there is another system, which may be designated as the "flocculus-stalk" bundle; this bundle is situated most ventrad and mesiad, is most extensive caudad and becomes less and less distinct as it ascends orad.

The intermedius bundle can be seen in all animals. It is situated most mesially in the nervus vestibularis and surrounds mesially the remaining portion of the bundle of the nucleus solitarius, the lateral boundary of which is the nervus vestibularis itself. This nuclear rest is not always distinct, but is best developed in the higher apes.

KESCHNER, New York.

ABORTIVE POLIOMYELITIS. EDITORIAL, J. A. M. A. **98**:405 (Jan. 30) 1932.

Since Wickmann first assumed the existence of an abortive form of poliomyelitis, the impression has become general that much, if not all, of the adult immunity to this infection is due to mass immunization of growing children by unrecognized subclinical attacks of the disease. Doubt as to the validity of this conclusion is implied in the current epidemiologic studies by Drs. Kramer and Aycock of Harvard University Medical School. While the Harvard investigators have not yet abandoned the conventional assumption that adult resistance to poliomyelitis is due to childhood contact with this specific virus, they seem to have been forced to the conclusion that effective contact with the immunizing virus does not take place at the time of epidemics. Apparently the immunization is a more or less continuous process extending throughout the year or perhaps even limited to interepidemic periods. Their data, however, are equally consistent with the recent radical European theory that the adult immunity is not so much due to specific contact with the infectious agent as to a nonspecific "serologic ripening" or "physiologic maturation" roughly synchronous with the development of sexual maturity.

A favorable opportunity for the study of abortive poliomyelitis was furnished about a year ago by a local outbreak of infantile paralysis in Bedford, Mass. (population, 1,700). With the appearance of the first case, the Boston investigators established residence in this suburb, where they remained for six weeks, made a house to house canvass and collected data from all mild illnesses dating from not more than two weeks preceding the first paralytic case. Approximately fifty such mild illnesses were studied. These illnesses usually consisted of unexplained headache, fever and vomiting, lasting from twenty-four to forty-eight hours. Thirty-three of these cases occurred among school children under 15 years of age, all cases of frank poliomyelitis developing in the same school.

About five months after this initial social survey, at which time any immunity resulting from the apparent abortive attacks logically would have developed, serums were drawn from about half of these school children, the pupils being selected to include every age group. As controls, serums were drawn from twenty-eight

local children of the same ages who had not exhibited recognizable illness at or before the time of the epidemic and from twenty-nine children from a neighboring city which was free from poliomyelitis and had remained so. Neutralization tests were made with the seventy-seven resulting serums. Within the limits of experimental error, the results were identical with the serums of the three groups. With the apparent abortive cases, 40 per cent of the serums neutralized poliomyelitis virus *in vitro*. In the two control groups 44.8 and 46.4 per cent, respectively, were demonstrably virucidal. A 41.9 per cent neutralization by serums of the same age groups was the average normal as determined by previous surveys.

Assuming that this neutralization *in vitro* is a dependable quantitative measure of individual specific immunity, the Harvard investigators were forced to the conclusion that "the widespread immunization of a population does not take place entirely at the time of an outbreak, but rather in a more or less uniform manner throughout the year, or in interepidemic periods." They do not draw the conclusion that this apparent continuous immunization is necessarily specific or that it is necessarily due to individual contact with the specific virus. They leave the questions of the specificity and genesis of the immunity open to further investigation. Of course, if future research should show that the virucidal power of serum is not a quantitative measure of individual resistance to poliomyelitis, their argument is invalid.

[EDITOR'S ABSTRACT.]

THE PSYCHOSEXUAL DEVELOPMENT OF THE CHILD. GERALD H. PEARSON, Ment. Hyg. **15**:685 (Oct.) 1931.

In the creation of behavior problems, the rôle of infantile maladjustment must be properly emphasized. Birth itself is a trauma, resulting in the sudden appearance of many unpleasant stimuli—noise, cold, dryness, carbon dioxide, etc. Only through the mother does the child find relief from these factors, and to its mother all its libido is attached. When the mother leaves the child, anxiety ensues. When the breast is withdrawn from the nursing infant, it fears that it will never be returned; in this manner, primitive anxiety states are brought into being, and at this time may be laid the basis of subsequent maladjustments. The child whose nursing has been curtailed will always feel cheated, always slighted; he will be the sort who considers himself consistently "picked on." Overnursing, too, results in problems; this child learns to expect everything and becomes irresponsible.

Pearson warns against attempting control of the bowel and bladder before the pyramidal tracts are developed, believing that demanding more of a child than he is physically capable of is psychically harmful. Training in excretion is an important process; by its overemphasis the parent will focus attention on the genitalia. Whatever control the infant does acquire is largely an effort at pleasing his mother; at first, in her absence, control is impossible, but later her memory image is enough to stimulate the child to control.

In lucid terms Pearson outlines the mechanism of the Oedipus complex and, in much less clear expressions, describes the development of the castration complex. He discusses sibling relationships, pointing out the special problems of the oldest child, the newest born, the middle child, and the only one. For example, he suggests that quarreling among brothers is due to the symbolization of the oldest boy as "father," and the privilege of venting the Oedipus-induced jealousy on him instead of on the real parent is irresistible.

Pearson analyzes the factors hindering psychosexual development. The triple mechanism of the ego, the id and the super-ego is in an easily disturbed balance; unusual stress, whether through trauma or training, on any one of these will upset the proportion. An overdeveloped super-ego may so suppress the manifestations of the id as to result in abnormal libidinous outlets. Tendencies to overreact to the stimulation of nongenital erogenous zones, such as the mouth or anus, may establish the foundation for an adult perversion. Id tension may

be released in one of three ways: (1) direct expression, (2) indirect expression, (3) changed expression. As an example of indirect expression, Pearson cites the nonsexual curiosity of children—curiosity about science, for instance, as a surrogate for direct but forbidden sexual curiosity. As an example of changed expression, the author suggests that the energy that drives scientists to painstaking study is but the modification of a primitive impulse of cruelty—or a desire to tear things apart.

The paper is closed by the presentation of two clinical examples.

DAVIDSON, Newark, N. J.

THE PERIVASCULAR LESIONS OF BORST IN MULTIPLE SCLEROSIS (HYPERLYMPHOSIS OR STATUS DISINTEGRATIONIS). MIKIO MURATA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **33**:211, 1931.

Murata cites Borst, according to whom little attention has hitherto been paid to the rôle played by disturbances of the lymphatic circulation in the pathogenesis of multiple sclerosis. Borst has repeatedly emphasized the fact that on account of the chronic inflammatory process in the meninges in this disease, the meningeal lymph spaces may become the site of lymph stasis. The accummulations of lymph are produced by adhesions in the adventitial lymph sheaths of the blood vessels and distention of the perivascular spaces by the glial proliferation. These are the so-called "Borst" lesions. They are arranged around the vessels, and consist of a disintegration of the glial supporting tissue, dilatation of the meshwork in the interglial spaces, swelling of the glia cells and more or less extensive destruction of the myelin sheaths in the presence of strikingly well preserved axis cylinders. The glia is also fibrous, and the entire process may become sclerotic.

Borst has also pointed out that the appearance of these foci differs in the spinal cord from that in the brain, and that not infrequently they are confused with secondary degenerations. He differentiated the secondary degenerations by their greater diffuseness and by the absence of a perivascular arrangement. Borst also emphasized that the lymph stases play only a secondary rôle in the pathogenesis of multiple sclerosis. He believed that the most important and primary pathogenic processes occur in the blood stream, and that the stasis of the circulation of the lymph is primarily due to changes in the blood vessels.

Such foci occasionally develop in association with the typical lesions of multiple sclerosis. In some cases Borst foci may originate not only from the margin of the cord, or from the larger vessels of the septum, but also from the sclerotic foci themselves; i. e., as the formation of a typical multiple sclerotic patch is coming to a gradual standstill there occurs a process of perivascular disintegration. On the other hand, a typical multiple sclerotic patch may also develop from one of these foci of perivascular disintegration; this, however, is a rare occurrence.

Murata examined histologically twenty-six cases of acute and chronic multiple sclerosis. Although many Borst foci were observed in most cases, there were some in which none were seen. There was a greater tendency to the formation of these lesions in the spinal cord than in the brain. Curiously enough, the brain stem contained less Borst foci than the cerebral cortex. This, he believes, is significant because it would seem to indicate that the process began in the meninges. Unfortunately, the meninges could not be examined in most of the cases that were studied. It is also noteworthy that in these areas of perivascular disintegration the destruction of the myelin sheaths is the primary and the glial proliferation the secondary factor in the production of the foci. In this sense, the process resembles closely that observed in perivascular disintegrations in senile brains.

KESCHNER, New York.

CISTERN PUNCTURE. LEO JACOBI, Am. J. Syph. **15**:544 (Oct.) 1931.

In a lengthy, detailed and well illustrated paper, Jacobi discusses the anatomy, indications and technic of cisternal puncture. He has nothing but praise for the procedure, pointing out in glowing terms the simplicity, safety and clinical value of cisternal puncture. He insists that it is less formidable than a spinal tap: that the patients are less likely to have a reaction, and that the procedure as a whole is much safer. In reviewing the history of cisternal puncture, he gives credit to Obreggia, who performed the pioneer tap in 1908, and deplores the priority commonly assigned to Ayer for his work in 1919. For finding the point of entry there are two methods, the Eskuchen and the Ayer. The former, or indirect method, uses the posterior edge of the foramen magnum as a point of orientation; the needle is inserted there, pointed a little above the meatus, and then depressed slightly to enter the atlanto-occipital membrane. In the direct (Ayer) method the needle is introduced in line with the upper edge of the auditory meatus or glabella until the dura is pierced.

As in the lumbar puncture, the choice of two positions is offered: horizontal and vertical. The recumbent position favors the spontaneous flow of spinal fluid because of the greater intracisternal pressure in that position; on the other hand, favorable flexion of the head can be maintained better in the upright posture. Jacobi suggests applying to the neck the compressor of Benedek and Thurzo, which is a sort of truss pressing on the jugulars. With this instrument the intracisternal pressure is so elevated that spontaneous flow occurs in the upright position. The rate of fatality is small—seven fatalities in ten thousand cases, including several fatalities in the early days of cisternal puncture. Jacobi believes that the likelihood of injuring the medulla is exaggerated, and that the danger of such an injury is less serious than might be expected. Among the complications he lists pain in the face, arm or leg, vomiting, hemorrhage and puncture of the bulb. These are all rare, and the only grave complication is hemorrhage into the cistern, the effects of which are prompt; most deaths attributed to cisternal puncture followed hemorrhage. If no more than 0.5 cm. of the needle enters the cistern, this complication is rendered most unlikely. Convulsions have occasionally been reported, but as these have occurred before the needle reached the dura the author suggests a psychic cause.

The contraindications to cisternal puncture are few; infants under the age of 3 are poor subjects because of the smallness of the parts; arteriosclerotic persons are not good risks because of the danger of hemorrhage. Choked disk or other evidence of high intracranial pressure is likewise a contraindication. Cisternal puncture has both diagnostic and therapeutic values. For diagnosis it is employed when the lower end of the spinal subarachnoid is blocked (as in meningitis when the lumbar tap needle becomes clogged with pus), as a method of introducing iodized poppy seed oil in taking a roentgenogram and as a means of localizing spinal block. Therapeutically, it is an excellent method of draining (encephalitis, uremia) and of introducing medication.

DAVIDSON, Newark, N. J.

PARINAUD'S SYNDROME WITH EXAGGERATION OF THE TONUS REFLEXES IN A CASE OF HEMORRHAGIC POLIENCEPHALITIS. E. FAÇON, A. BRUCH and S. CHISER, Rev. d'oto-neuro-opht. **9**:575 (Oct.) 1931.

The association of disturbances of synergic motility of the ocular globes with hemorrhagic poliencephalitis is rare. In such cases Grasset, Parinaud and Cantonnet postulated a superior center of coordination for oculogyric movements and convergence, located in the vicinity of the oculomotor nuclei.

A man, aged 49, entered the clinic complaining of difficulty of speech and of movements of the right limbs and eyes. For the previous nine years he had had diabetes, which was well controlled. Six weeks before admission he had a sudden attack, characterized by vomiting, profuse sweating and a poor general condition. Examination revealed fixed gaze, immobile facies, slight dysarthria, abolition of the movements of raising, lowering and converging the eyes, facial asymmetry,

right lagophthalmia and nystagmus. A vestibular test showed marked labyrinthine hyperexcitability. There were difficulty in executing fine movements of the fingers of the right hand and diminution of the segmental force in the flexors of the right leg. The tendon reflexes were more lively on the right side; there was a bilateral Babinski reaction; standing and walking were difficult, with a tendency to fall to the right, and there was dysmetria in the right arm and in both legs. The spinal fluid was xanthochromic; the tension was 15 (Claude) in the lateral decubitus; the Pandy and Nonne-Apelt reactions were positive, and the lymphocyte count was 8 per cubic millimeter. The Bordet-Wassermann reaction was negative. The blood sugar was from 12 to 15 mg. per hundred cubic centimeters, and the urine contained 5 mg. of dextrose per hundred cubic centimeters. After being in the hospital for a month, the patient was improved, but convergence was still impossible. With the arms extended, the right hand tended to pronate, and the arm deviated downward; when the head was rotated to the right, the right arm deviated markedly to the right, but there was little deviation of the arm on turning the head to the left.

These phenomena are a part of a group of tonic reflexes that preserve the static equilibrium of the body. These reflexes appear when the tonus centers (red nucleus, vestibular nuclei and centers in the cervical cord) are freed from the restraining action of the cerebellum. Marinesco and Radovici have published a case of tetraplegia with generalized rigidity, resembling that seen after decerebration, in which the tonic cervical and labyrinthine reflexes on the extremities were demonstrable. In the present patient, it is probable that the inability to converge the eyes is related to the appearance of the tonus reflexes; the limitation of movements of the globe toward the inner orbital angle is merely an expression of a superior tonic phenomenon, analogous to the other reflexes that were found.

DENNIS, Colorado Springs, Colo.

THE DIFFERENTIATION OF PERIPHERAL ARTERIAL SPASM AND OCCLUSION IN AMBULATORY PATIENTS. W. J. MERLE SCOTT and JOHN J. MORTON, J. A. M. A. **97**:1212 (Oct. 24) 1931.

Scott and Morton outline a simple method to differentiate vasoconstrictor spasm from occlusion in the common vascular diseases of the extremities. This test can be carried out easily in ambulatory patients. It has eliminated the inconvenience and danger of previous methods and does not require hospitalization. In performing the test on the lower extremity one ascertains by the accepted methods of clinical examination that a circulatory deficiency exists in it. The patient is placed at rest on a bed or table, with the feet and legs to the knees exposed, in a quiet room in which the temperature (at the level of the patient) is as near as possible to 20 C. (The range permitted is from 18 to 23 C.) The temperatures on the toes and soles are taken at five minute intervals with an instrument which accurately measures the true skin temperature. After the level of the surface temperature has been established (the surfaces are usually exposed at least twenty to thirty minutes), the posterior tibial nerve is blocked just below the internal malleolus, by the injection of from 8 to 10 cc. of 1 per cent procaine solution about it. If the injection is properly made, loss of tactile sensation in the area of distribution begins within fifteen minutes. In order that this test shall be valid, complete anesthesia to touch must be obtained in the area investigated. The surface temperatures are recorded at intervals (every five minutes) until the maximum is attained; this occurs usually from fifteen to thirty minutes after the induction of a satisfactory anesthesia. The height of the surface temperature in the anesthetic area is then corrected for the room temperature; 0.3 degree is added to or subtracted from the reading for each degree by which the room temperature exceeds or falls below 20 C. This corrected maximum is then compared to the normal vasodilatation level. If the surface temperature of the toe has gone up to equal or exceed 30.5 C., vasoconstrictor spasm is the cause of the local circulatory deficiency. If there is no increase in the temperature following the nerve block, vasoconstriction plays no rôle in the vascular disease. And if there is a definite

increase in the surface temperature, which fails, however, to reach the normal vasodilatation level, the condition belongs in the mixed group and vasoconstriction is an element but not the only element in causing the circulatory deficiency. This method of differentiating the effects of vasoconstriction from those of occlusion is applicable also to the upper extremity. The ulnar nerve at the elbow or the median nerve just above the wrist is blocked with procaine and the temperature changes in the anesthesized area are followed. If the hand is cool initially, the hyperthermia induced is so striking that the difference in temperature between the two sides of the ring finger has easily been appreciated by palpation during ulnar anesthesia. The normal vasodilatation level is approximately the same as for the feet.

EDITOR'S ABSTRACT.

THE RESPONSE OF A MUSCLE SPINDLE DURING ACTIVE CONTRACTION OF A MUSCLE. BRYAN H. C. MATTHEWS, J. Physiol. **72**:153 (June 26) 1931.

A previous paper by Matthews described a series of experiments on the behavior of a single stretch receptor in a frog's muscle; in these experiments the response of the end-organ during passive stretching was investigated by observing the action potentials in the nerve supplying this muscle. The present paper describes a further series of experiments made to determine how these end organs respond when the muscle contracts actively. The single end-organ preparation from the frog's toe described in the previous experiments was again utilized.

If the muscle is under slight initial tension and twitches either isometrically or isotonically there is little sensory response during the rising phase of the mechanical response, but as relaxation occurs there is a considerable discharge of impulses. There is little or no sensory response if the muscle is slack. It therefore appears that the action current of the muscle does not stimulate the muscle spindle. If the muscle is under some initial tension, when it contracts either isometrically or isotonically, the sensory response to that tension ceases during the rising phase of the mechanical response, and restarts at a higher frequency during relaxation. The pause in the sensory response to stretch, which occurs when the muscle retracts, lengthens with the mechanical response when the muscle is fatigued; it seems to be connected only with the mechanical events in the muscle. During tetanization there is no response from the muscle spindle; if the initial tension is considerable the sensory response to that tension ceases during tetanic contraction of the muscle, and restarts, often at a higher frequency, when the muscle relaxes. The pause also occurs in the sensory response from muscles containing a number of stretch receptors. In some preparations, increasing the strength of the shock applied to the nerve obliterates the pause. It is suggested that this is due to contraction of the terminal cross-striated portions of the muscle spindle. These have a separate motor fiber, and this motor fiber seems to have a higher threshold than the ordinary motor fibers to the muscle. The intrafusal fibers also seem to have a higher threshold than the other muscle fibers.

Impulses descending the nerve to the muscle spindle evoked by electrical stimulation set up a refractory period in the nerve ending. There is no evidence of a compensatory pause. Descending impulses reaching the nerve ending at a frequency of 100 per second appear to reduce its adaptation. The pause in the response of the muscle spindle to steady tension when the muscle contracts is compared to the silent period in mammalian reflexes. The author suggests that the silent period is due to the pause in the response from the muscle spindles.

ALPERS, Philadelphia.

OXYCEPHALY AS A PATHOGENETIC ENTITY. A. N. BRONFENBRENNER, Am. J. Dis. Child. **42**:837 (Oct.) 1931.

The author endeavors to portray oxycephaly in terms of a pathogenesis rather than from the angle of its usually described complication, optic neuritis. It is interpreted in the majority of textbooks as a deformity of the skull with certain morphologic characteristics. An unusually high head and steeply rising forehead

constitute its outstanding features. They tend to fall into the brachycephalic group. The eyes are more or less protruded, and strabismus and nystagmus are typical findings; atrophic changes in the optic nerve are intimately related to the condition. Mental defect of a mild degree occurs in a certain number of reported cases.

Bronfenbrenner reports five cases, in all of which there was a history of persistent headaches and progressive loss of vision. In four of the five cases there was a positive Pirquet reaction and a history of tuberculosis in the parents of one patient. All five of the patients were subnormal in their mental development. The intelligence quotients varied between a low of 33 and a high of 75. Roentgen studies of the skulls of all showed sequelae of increased intracranial pressure, such as a premature synostosis of all sutures of the skull and a tendency to craniostenosis as demonstrated by marked convolutional digitations, attentuation of the calvarium, narrowing of the diploe and flattening of the air sinuses. All of the cases showed a disproportionate height of the skull as the one consistent morphologic feature. The patients presented different degrees of optic atrophy, the majority being of the postneuritic variety, but they demonstrated that atrophy of the optic nerve was not an indispensable sign of oxycephaly. The author considers oxycephaly as a compensatory adjustment mechanism, as an effort of the individual to meet the demands of a growing brain in a skull with prematurely closed sutures. The increased intracranial pressure that follows the premature synostosis naturally damages the brain, especially the very sensitive optic nerve, and retards the development of the higher intellectual faculties. He believes that the premature synostosis may be a sequel of a chronic meningitis, especially of a low grade tuberculous or syphilitic type, particularly as four of the patients gave positive von Pirquet reactions and a history of tuberculosis in the parents. He reports instances of three cases of syphilitic oxycephaly in one family and in two of his own patients. Only one of his cases presented any signs of lymphatic dyscrasia and rickets, despite the high incidence of rickets that had previously been reported as an etiologic factor by other authors.

The cases are presented in a clear, concise and descriptive manner, and constitute a definite and helpful addition to the rather scanty literature on this subject.

LEAVITT, Philadelphia.

OBSESSIVE PHENOMENA IN EPIDEMIC ENCEPHALITIS. D. S. OZEREZKOVSKY and M. A. DSAGAROV, Rev. psychiat., neurol. & reflexol. (Leningrad) **5**:6, 1930.

There is an extensive literature on various neurotic symptoms, including compulsive phenomena and obsessions, in connection with encephalitis. The subject has been covered extensively in the German literature, especially by Runge. The authors report three cases of postencephalitic parkinsonism, with the usual sequelae of rigidity, increased salivation, immobility and other vegetative disturbances. In the first case, a girl, aged 23, had an acute attack of encephalitis in 1923. A year later, she was admitted to the hospital, where she began to complain of evil thoughts, which usually appeared when she was praying. Later on, she began to complain of constant sex thoughts and of an uncontrollable desire to choke somebody. This compulsion was so strong that on several occasions she attacked nurses, patients and relatives. She was often distressed by the appearance of disconnected thoughts. She began to feel that if she screamed certain words she would be more comfortable. The patient had excellent insight, but she could not control her thoughts, and was so distressed by them that she attempted suicide several times.

In the second case, a boy, aged 20, who entered the hospital in 1927 and was discharged nine months later, had had acute encephalitis, with parkinsonian symptoms appearing a few months later. The patient was especially distressed by oculogyric crises during which all kinds of thoughts came to his mind. He had a compulsion to disjoin words into syllables and then to combine them together, giving rise to meaningless words and sentences, with a tendency to read them in a reverse order. Compulsions to hit the physicians and nurses appeared. He began to complain of sex tension, and asked the staff to arrange some contact

with the female wards so that he could satisfy his desires. The compulsive ideas disappeared after a few hours of sleep and then reappeared. The patient had good insight, and was distressed by his condition.

In the third case, a man, aged 36, gave a history of having had an acute attack ten years previously. In this case, the obsessive phenomena expressed themselves in an uncontrollable desire to touch women. These attempts led finally to the loss of his position and numerous conflicts with the police.

As the symptoms in the three cases were not different from those in typical cases of obsessive neurosis, the author states that there is no reason to call them anything else. Although the content of the patient's psychic life is determined by his experiences, the peculiar dynamics by which the patient's conflicts express themselves are due to cortical and subcortical mechanisms. The involvement of the basal ganglia releases certain motor phenomena which express themselves in compulsive thoughts.

KASANIN, Howard, R. I.

AN INTERESTING CASE OF MEGALENCEPHALON. ISIDORO IMBER, Rassegna di studi psichiat. **20**:749 (July-Aug.) 1931.

The author reports an interesting case of megalencephalon, in which the brain was exceptionally heavy, in the absence of tumor or of hydrocephalus. The patient, an idiot, aged 24, died of pulmonary tuberculosis. The brain and the cerebellum together, without the dura, weighed 2,633 Gm. The cerebellum was not developed in proportion, and weighed only 180 Gm. The convolutions were very large, and the sulci very deep. The thickness of the cortex was practically double the normal. No plaques of tuberous sclerosis or subcortical gliosis were present. With the exception of two small areas of softening, nothing of importance was noticeable in the basal ganglia. In the mesencephalon, the cells of the substantia nigra were greatly reduced in number, and those present were somewhat sclerotic and poorly stained. In the cortex, the Betz cells were larger than usual. There was a general slight hypertrophy of neuroglia cells. The author thinks that the destruction of the substantia nigra is an index of an encephalitic process.

The author recalls other cases in the literature of true macrocephaly, that is, megalencephaly, especially that of the heaviest brain, 2,850 Gm. in weight, reported by Walsen and Leunei, and those in which the brains weighed, respectively, 2,480 Gm., reported by Simms; 2,155 Gm., reported by Schmincke; 1,995 Gm., reported by Anton; 1,874 Gm., reported by Volland; 1,860 Gm., reported by Hensman, and 1,860 Gm., reported by Middlemas. The fact that there was a dysharmony between the development of the cerebellum and that of the brain is considered by Schluter and Peter as an occurrence among idiots, whereas a harmoniously overdeveloped brain and cerebellum are found in normal persons. Schmincke often found megalencephaly associated with acromegaly. In the case reported by Imber there were no pituitary disturbances.

Megalencephaly may be of two types: (1) that in which hypertrophy is proportionate in all nerve elements (true hypertrophy), and (2) that in which there is an increase of the interstitial tissue (hyperplasia). If for the cases of interstitial hyperplasia a theory might be advanced of a difficulty in the development of the nerve cells and nerve fibers in an early embryologic period, this theory cannot be applied in cases of true hypertrophy, as in the author's case. In the cases of true hypertrophy, it is more plausible to admit a strength of evolution of embryonal tissue superior to normal. It remains, however, to be seen if normal embryologic tissue may acquire evolutionary powers superior to normal when under the influence of exogenous factors.

FERRARO, New York.

DERMATOMYOSITIS. G. MARINESCO, ST. DRAGANESCO and E. FAÇON, Ann. de méd. **30**:144 (July) 1931.

A chauffeur, aged 18, entered the hospital with complaints of pain, difficulty in the use of both the upper and the lower extremities and difficulty in swallowing. The left cheek, the nose, the eyebrows and the upper part of the face were

swollen, red and painful to pressure. The outlines of the swollen parts were diffuse, with desquamation of the surrounding skin. There was difficulty in swallowing fluids; the speech was slow and nasal. Movements of the upper jaw, and of the upper and lower extremities were carried out with difficulty; the muscular power was diminished. The muscles of the arms and hips were diffusely swollen, hard and painful to pressure. Tendon and cutaneous reflexes were normal; sensation was not disturbed. Electric examination of the muscles involved showed a quantitative diminution of reaction to faradic and galvanic stimulation. Histologic examination of a piece of muscle removed at biopsy showed an intensive interstitial inflammatory reaction, with degeneration of the muscle fibers.

The patient died after six months. Microscopic examination showed multiple inflammatory foci of the subcutaneous tissue, the interstitium and the parenchyma of the different muscles involved. The cellular infiltration consisted mainly of lymphocytes, together with a marked proliferation of sarcolemma nuclei. In the peripheral nerves, isolated small foci of demyelinization were found, with isolated degenerated nerve fibers. The most prominent feature was the infiltration of small round cells in the perineurium and around the interstitial vessels. A similar picture was seen in the capsule of the spinal and sympathetic ganglia. In the dorsal spinal cord, minute lymphocytic cuffing around smaller veins of the lateral horns and the white matter was found.

"Poikilodermie myopathique" (type Petges-Clejan) is closely related to dermatomyositis. The pigmentation of the skin in the first disease and the spreading of the inflammation to the corium are important points of differential diagnosis. Sclerodermia presents a histologic picture similar to dermatomyositis, but the chronic course and the typical cutaneous lesions characterize the first disease. In polyneuritis, cutaneous changes are absent. Kussmaul's disease is recognized by the periarteritis nodosa. Tuberculosis, rheumatism, angina, influenza, focal sepsis, etc., have been considered as etiologic factors. The authors think of a filtrable virus, similar to that which had been demonstrated in periarteritis nodosa, with a special affinity for connective tissue and vessels.

WEIL, Chicago.

THE MENACE OF PSYCHIATRY. J. BRENNEMANN, Am. J. Dis. Child. **42**:376 (Aug.) 1931.

Considerable comment has been received in neurologic circles regarding this address of Dr. Brennemann before the New England Pediatric Society. In his paper he adopts the attitude of "The Pediatrician Views Psychiatry," at times mentioning the various ramifications of psychiatry with a delicate sarcasm, and at others contrasting psychiatric dogma with pediatric experience. He particularly flays the "behaviorism" of Watson and quotes him to some extent. His handling of the problem of the "I. Q." corresponds very much with the attitude of the older school of psychiatrists in that they lay stress on the quotient and more on an individual evaluation of the child as a whole. He compares the attitude of the psychiatrist and of the pediatrician toward the child patient, and confesses that in psychiatric disorders the psychiatrist has a more profound theoretical knowledge of the subject and a deeper insight into the condition than has the pediatrician. On the other hand, he thinks that in the majority of the psychologic situations that develop in the growing child, the pediatrician is better qualified to evaluate them because of his greater experience in dealing with children from infancy to adolescence and by his more intimate and familiar contact with the normal or abnormal child who has not yet become an object of psychiatric investigation. He refers to the danger in the wholesale education of the laity in all that pertains to child study, guidance, behavior training, psychology and psychoanalysis. He does not believe in the present vogue of educating the parent in all of the psychologic and psychiatric problems of childhood, because of the danger of false diagnosis under the old theory that " a little knowledge is a dangerous thing." In conclusion, he suggests that psychiatrists declare a truce or moratorium

in their present tendencies, and that they investigate their present psychiatric movement as a problem of its own. His interpretation of the "Menace of Psychiatry" is that the too intensive injection into the lay mind (which cannot assimilate it) of a mass of complicated ideas in a domain that requires wary walking will cause more harm than good. In other words, he thinks that psychiatrists in their efforts to meet a problem are only creating more problems.

As an after-dinner talk or presidential address the article is extremely interesting, but it contains no new facts or ideas. It is of value in that it indicates the attitude of the older school of pediatricians toward the ramifications of the newer psychiatry.

LEAVITT, Philadelphia.

BRAIN ABSCESS TREATED BY THE METHOD OF EXCLUSION OF THE SUBARACHNOID SPACES. AUBIN and E. BAUDOUIN, Rev. d'oto-neuro-opht. **9**:582 (Oct.) 1931.

One more successful result from the method of treatment for brain abscess by exclusion of the subarachnoid spaces is added to the number previously reported.

A man, aged 40, who had had otorrhea in the right ear since childhood, was in a precarious condition when first seen, with marked contraction and stiffness of the neck, profound coma, a temperature of 40.5 C. (104.9 F.) and a pulse rate of 130 per minute. No diagnosis other than generalized meningitis was possible. Immediate operation was undertaken. The spinal fluid, taken immediately before the intervention, was clouded, and no micro-organisms were found on direct examination. When the roof of the antrum was removed, a large extradural abscess was discovered, and the meninges were red and fungoid. Puncture of the temporal lobe revealed two coffeespoonfuls of greenish-yellow, fetid pus. A small rubber drainage tube was substituted for the needle, and the wound was dressed. The pus contained streptococci and gram-positive and gram-negative anaerobic bacilli. Improvement was immediate. The drain was increased progressively in size at each dressing. On the fifth postoperative day an intercurrent infection of the wound caused a rise of temperature. Reexamination of the patient revealed: The stiffness of the neck was still present; the sign of Brudzinski was positive; there was a flaccid right hemiplegia, more marked in the arm, and Babinski's sign was positive. The face was not involved in the paralysis; sensibility was not disturbed, and there were no ocular abnormalities. A striking change in the psychic state of the patient occurred. He had no interest in himself or his surroundings, although his comprehension was perfect. Ordinarily taciturn, he became loquacious, and his conversation was accompanied by immoderate, puerile laughter. There was no interruption of the favorable course of the healing, and the cure was complete in seven weeks. Five months after the operation he was able to resume his occupation.

Attention is called to the unusual seat of the hemiplegia, the signs of purulent meningitis, the psychic alterations, analogous to those seen in patients following an operation for cerebral tumor, and the advantages of the method used for draining a brain abscess.

DENNIS, Colorado Springs, Colo.

TUBERCULOUS MENINGITIS WITH SYPHILITIC MENINGITIS TERMINATING IN RECOVERY. DAVID W. KRAMER and B. B. STEIN, Arch. Int. Med. **48**:576 (Sept.) 1931.

The popular idea that tuberculous meningitis is necessarily and invariably fatal is condemned by Kramer and Stein, who find reported in the literature over 400 cases of this disease with recovery. They present a patient, aged 29, who had contracted syphilis eight years before admission to the hospital. His chief complaint at the time of admission was headache; he also suffered from vertigo, diplopia and vomiting. He had irregular Argyll Robertson pupils, strabismus, rigid neck, positive Kernig sign, optic neuritis and signs in the upper part of the right side of

the chest suggestive of pulmonary tuberculosis. A first spinal tap revealed cloudy fluid under normal pressure, with a cell count of 1,200, including 44 per cent of lymphocytes. The colloidal gold curve was of tabetic form, and the Wassermann reaction of the spinal fluid was 4 +. Sodium iodide was administered intravenously, and mercury was given by inunction. Ten days later, clear spinal fluid, in which a pellicle formed, was obtained. At this time the Levinson reaction was positive. Tubercle bacilli were found in the pellicle. The fluid was inoculated into a guinea-pig, which died three months later; no tubercles were found in the animal. The authors believe that their patient had syphilitic meningomyelitis, which, because of its acute onset, was probably not tabetic. They believe that the iodides broke down a tuberculous focus in a lymph node, freeing bacilli which then entered meningeal tissue already weakened by syphilis.

That the organisms were found only once may, Kramer and Stein confess, cast some doubt on the diagnosis. There was, however, no doubt that the bacteria actually were tubercle bacilli. The failure to reproduce the disease in a guinea-pig may have been due to the involvement by a strain of low virulence or to the small number of organisms in the body. In analyzing the cases of tuberculous meningitis in the literature in which recovery was reported, the authors find a correlation between age and prognosis which suggests that the older the patient, the better are his chances of escaping the usually fatal termination. In view of the possibility of recovery, Kramer and Stein suggest that the family of a patient with tuberculous meningitis be encouraged by the admission of slight hope.

The literature on prognosis in this disease is carefully reviewed and clearly tabulated.

DAVIDSON, Newark, N. J.

OTOGENOUS ENCEPHALITIS OF THE NONEPIDEMIC TYPE. G. V. T. BORRIES, Rev. d'oto-neuro-opht. **9**:557 (Oct.) 1931.

In 1921, Borries called attention to the possibility of a nonsuppurating encephalitis, complicating otogenous infections, as a distinct entity, apart from cases in which the condition is a part of the process in cerebral abscess. As examples of the condition, he cited such experiences as the following: 1. In a patient with an infection of the ear aphasia and hemiplegia develop; lumbar puncture and cerebral puncture give negative results, and the patient gets well. 2. In a patient who dies with a cerebral complication of an infection of the ear, autopsy reveals a large encephalitic lesion but no pus; such a condition cannot be considered as the initial stage of a cerebral abscess. In both cases the appropriate diagnosis is nonsuppurating otogenous encephalitis.

For the most part the symptoms correspond to those of cerebral abscess, and differentiation is difficult. The importance of recognizing the possibility of the existence of this complication is illustrated by three clinical pictures: 1. In a patient in the acute stage of otitis media who exhibits cerebral symptoms, it must be determined whether there is present an epidemic type of encephalitis or an otogenous complication. If the latter, it must be remembered that nonsuppurating otogenous encephalitis may exist, even with negative results from lumbar or cerebral puncture. 2. In a patient with influenzal or epidemic encephalitis, suppurating otitis and cerebral symptoms, doubt exists as to whether or not there is an otogenous complication; if cerebral puncture gives a negative result, it does not exclude the possibility of an inflammation of the brain of otogenous origin. 3. In a patient with otitis media on the left side aphasia and hemiplegia develop; there is no meningitis, and cerebral puncture gives negative results. If the possibility of otogenous encephalitis is not considered, repeated attempts to locate an abscess might be made, to the detriment of the patient. If recovery occurs, the surgeon is not able to explain the case unless the hypothesis of nonsuppurating encephalitis of otogenous origin is taken into account.

DENNIS, Colorado Springs, Colo.

OBSERVATIONS ON THE STRUCTURE OF THE SURVIVING NERVE CELLS IN ORTHOPTERA. G. BRUNO, Riv. di neurol. **4**:363 (Feb.) 1931.

In order to demonstrate the existence of neurofibrils in the living nerve cell, which has been denied by previous workers, such as Pensa, Ettisch and Jochims, the author, immediately after decapitation of Pachytilus migratorius, Acridium rineola, and other Orthoptera, studied the brain and subesophageal ganglia in a juice furnished by the animal under observation. The author followed the histologic structure of the nerve cells from the time of death to several hours after death, and reached the conclusion that in the cerebral ganglia as well as in the thoracic ganglia of Orthoptera the nerve cells are formed by fine granular cytoplasm in which granules of various dimensions and probably of different nature are located. This cytoplasm possesses, because of its viscosity, a considerable plasticity and resistance. The small granules in the cytoplasm surround the nucleus concentrically.

The cytoplasm of the nerve cell is surrounded by a pericellular, nucleated capsule with a syncytial structure adhering intimately to the cell body and passing directly into the nerve process. The capsule detaches itself from the cell and from the processes when water from the surrounding media penetrates between the capsule and the cytoplasm.

The cytoplasm rapidly undergoes modification, and a reticular structure occurs which becomes more and more evident and better defined. This reticulum can be stained with methylene blue (methylthionine chloride) or toluidine blue. Conversely, the use of a solution of mercuric chloride, which coagulates the cytoplasm, causes the disappearance of the reticular structure and the appearance of small, granular masses in the cytoplasm. The cone of origin of the axis cylinder possesses delicate longitudinal striations which probably belong to the capsule and not to the cytoplasm.

The results of the author's investigation confirm, therefore, the absence in the living nerve cells of preexisting neurofibrils.

FERRARO, New York.

THE FUNCTIONS OF THE GLIA IN SECONDARY DEGENERATION OF THE SPINAL CORD; THE OLIGODENDROGLIA AS PHAGOCYTES. FRITZ CRAMER and BERNARD J. ALPERS, Arch. Path. **13**:23 (Jan.) 1932.

A study of experimental secondary degeneration of the fibers of the spinal cord in rabbits was made with the use of the metallic impregnation methods of the Spanish school, the aim being to study the three types of glia in this reaction. The degenerative reactions were studied in various representative stages up to and including the seventy-eighth postoperative day. The study revealed and confirmed the fact that the glia cells, both ectodermal and mesodermal, were the active agents in the phagocytosis of the degenerated nerve fibers. In conjunction with a belated activity on the part of the microglia, the oligodendroglia were shown to continue their phagocytic activities until the axomyeline material had been fermented into true fat.

The substantiation of the fact of active phagocytosis on the part of the oligodendroglia, accompanied by that of the microglia, was made on the basis of distinctly different cyto-architectonic qualities in the phagocytic derivatives of these two types of cells. The phagocytic-digestive activity of the ectodermal types, i. e., the oligodendroglia, is compared with that of glandular cells. The phagocytic digestive activity of the ectodermal types, i. e., the oligodendroglia, is compared with that of glandular cells.

The astrocytes were shown to have a particularly sturdy reaction during the earlier weeks of the degenerative process, with some exceptions, but in the later weeks to engulf fat and the other products of the degenerated nerve fibers. A hyperplasia on the part of the ectodermal glia, leading to the formation of new oligodendroglia and macroglia, presumably for the purposes of the ultimate

digestion and disposition of the fat, was demonstrated. The last function of the macroglia was seen to be in the nature of cicatrix formation in the areas of degeneration.

WINKELMAN, Philadelphia.

HARMALOL IN THE TREATMENT OF PARKINSONISM. H. ASTLEY COOPER and J. A. GUNN, Lancet **2**:901 (Oct. 24) 1931.

An alkaloid, harmine, obtained from Peganum harmala, has been used clinically in Germany by Lewin and Schuster since 1928. By the end of 1930, a considerable number of clinical reports had been published, which generally showed that harmine favorably influences the muscular rigidity in postencephalitic conditions, but that it has little or no effect either on the tremor or on the mental condition. Halpern explained its activity as being different from that of hyoscine, in that the latter has a depressant action on the extrapyramidal tract while harmine stimulates the motor cells of the cerebral cortex, thus facilitating the passage of voluntary impulses through the pyramidal tract. This explains the fact that harmine does not relieve the tremor, but produces a curious feeling of "lightness" of the muscles.

One of the authors and his co-workers have investigated some artificial derivatives of harmine. He found that while in all compounds investigated the actions on muscle were similar, there were important differences in their action on the central nervous system. Harmine produces convulsions in laboratory mammals, while harmol and harmalol (which differ from harmine in the substitution of HO for the CH_2O group) have no such convulsive effect.

The results obtained are as follows: Harmalol hydrochloride in doses of ⅛ grain (0.052 Gm.) hypodermically, or of from 8 to 10 grains (0.52 to 0.65 Gm.) by mouth, has slight but definite effect in relieving muscular rigidity in parkinsonism. In these doses it is well tolerated. It has no beneficial effect on the tremor or on the mental condition, and does not relieve salivation. It does not produce anything like so complete a relief of the symptoms as is effected by the alkaloids of the hyoscine group, but may be of some value as an adjuvant to these in the treatment for parkinsonism.

BECK, Buffalo.

MÉNIÈRES'S DISEASE. W. S. THACKER NEVILLE, Brit. M. J. **2**:54 (July 11) 1931.

The various theories of the etiology of Ménière's disease are considered. First, there is the older concept that it was due to hemorrhage into the labyrinth. Then follow the theories of intracranial causation, such as lesions in the medulla oblongata (Thornval), lesions of the auditory nerve (Charcot, Dandy), pressure on the nerve from fluid around it (Aboulker), increased intracranial hypertension acting on the otoliths via the ductus endolymphaticus (Quix) and increased pressure in the cisterna pontis (Kobrak). Intra-aural causes are next in consideration. There are the toxin theory (Witmaack), angioneurotic edema of the labyrinth (Quincke), "glaucoma of the labyrinth" due to hypertension of the labyrinthine blood vessels (Portmann) and a sympathetic effect on the vascular supply, suggested because cocaine applied to the sphenopalatine ganglia (Halphen) relieved the symptoms. Faulty water metabolism constitutes the last theory to be considered (Dederding, Mygind). Considerable evidence to support this contention is presented. Symptoms are interpreted as being due to an increase of pressure in the semicircular canals, which produces vertigo and deafness by displacement of the stapes.

The author advocates treatment based on this concept. An attempt must be made to dehydrate the patient, as by restriction of fluid intake, promotion of the loss of water by carbonic acid, fir needle, mud baths, Scotch douche or exposure to the carbon arc massage. The diet must be salt-free. Local treatment consists in catheterization of the eustachian tube. A patient with an acute attack is treated with atropine and ergotinine. An injection of salyrgan is followed by a long course

of calcium-theobromine sodiosalicylate, while calcium and ovarian and thyroid extracts are also used. The author also uses bulbocapnine (Merck); 0.1 Gm. orally, in daily doses, prevents vertigo, and 0.1 Gm. hypodermically will give relief from the acute attack almost at once. FERGUSON, Niagara Falls, N. Y.

RETINAL CHANGES IN HYPERTENSION AND IN RENAL DISEASE. ROLFE FLOYD, Arch. Ophth. **6:** 433 (Sept.) 1931.

This article is in the nature of an ophthalmologic review. It considers retinal changes in arterial hypertension and in renal disease. The article is classified under a group of subheadings: (1) The changes in the eyegrounds in general: In this subsection the author discusses the ophthalmologic picture of the pathology of blood vessels as seen in the various lesions—miliary aneurysms, perivasculitis, arterial thrombosis and obliteration, etc. Hemorrhages and the various types of exudates and changes in the nervehead are also considered. (2) The retinal lesions themselves: edema, hemorrhages, sclerosis of the retinal arteries, degeneration of the retinal tissues and the deeper lesions that appear. (3) Renal disease and hypertension: Here are considered the modern classification that divides renal lesions into three main groups—pathologic conditions of the tubular epithelium; pathologic conditions of the glomeruli and renal sclerosis. From an ophthalmologic standpoint, the second and third are alone important. In the first, as the reviewer states, the nephrosis group may quickly be disposed of, because in it hypertension is most unusual and changes in the eyegrounds have not been described, except in the presence of amyloid kidneys. He includes this latter condition in the first group of the classification under protest.

In the second subsection, the pathogenesis of the retinal lesions and the manner in which the various changes develop are discussed. In the last subsection, changes in the fundus in hypertension and in renal disease are correlated, i. e., the sclerosis of the retinal vessels, the hemorrhages, the various exudates that appear, the various types of neuroretinitis and the swelling and edema of the nervehead.

In closing the review, the diagnostic and prognostic significance of changes in the fundus are considered. Rather satisfactory consideration is given also to the prognostic opinions presented by various other authors.

SPAETH, Philadelphia.

ATYPICAL COURSE OF DEMENTIA PARALYTICA TREATED WITH MALARIA. M. M. MIRSKYA-GINSBURG, Rev. psychiat., neurol. & reflexol. (Leningrad) **5:**16, 1930.

In the German literature one finds an interesting description of a "paranoid-hallucinatory" syndrome in connection with the malarial treatment for dementia paralytica, which was described originally by Gerstman and subsequently confirmed by other investigators. This clinical phenomenon raises a number of interesting problems, such as the possibility of the transition of dementia paralytica into other forms of syphilis of the brain, the relationship of this "paranoid-hallucinatory" syndrome to various exogenous and endogenous factors, the relationship to schizophrenia, malaria as a possible source of the syndrome, the possibility of cerebral localization and the clinical course of cases complicated by the appearance of the syndrome. The author observed this syndrome in 14 of the 279 patients (5 per cent) treated in the Bechterew Brain Institute since 1924. A careful anamnesis showed no evidence of excessive alcoholism. The syndrome appeared a few days after inoculation with malaria, during the incubation period of the illness. There is a serious question as to the relationship of the syndrome to schizophrenia. However, the auditory, visual and kinesthetic sensations are so vivid that they resemble more the false sense perceptions that one finds in delirium rather than those in schizophrenia. The author postulates that the malarial infection itself is probably the cause of the hallucinatory state, which has all the

symptoms of a toxemia or an infection. The fact that the hallucinations appear with the paroxysm and disappear after the chill strengthens this theory. The delirium is caused by the malarial infection, which finds a ready soil in the pathologically altered nervous system of the patients with dementia paralytica.

KASANIN, Howard, R. I.

TEMPOROSPHENOIDAL ABSCESS ON THE LEFT SIDE, OF MASTOID ORIGIN. DRAINAGE BY LEMAITRE'S METHOD. CASTELNAU, Rev. d'oto-neuro-opht. **9**: 563 (Oct.) 1931.

This case report concerns a woman, aged 45, who had had a suppurating left ear since childhood. Seven days before the consultation, an acute lighting up of the infection occurred, accompanied by persistent headache and occasional attacks of vertigo and otalgia. After another week of observation, an operation on the mastoid was done, but the progress of healing was unsatisfactory. The headaches persisted; the temperature remained above normal, and the wound was filled with unhealthy granulations. No clear signs of intracranial involvement were elicited, and the eyegrounds and spinal fluid were normal. On the ninth postoperative day, the patient suddenly became worse: The lassitude increased, vomiting recurred, and the psychic state was changed, but no other evidence of intracranial mischief was apparent. The urine was scanty; urea was found in the spinal fluid and was increased in the blood. During the night, coma developed. Exploration of the temporal lobe revealed an abscess containing 5 cc. of pus. The abscess was drained by the method of Lemaitre.

The evolution of the abscess probably began in the first days of the acute exacerbation of the mastoiditis. The course of the disease was modified by the partial decompression incident to the first operation. Atypical mastoiditis should suggest the presence of cerebral abscess, and persistent headache is a valuable sign, especially when accompanied by change of character and lassitude, even in the absence of torpor, obnubilation and aphasia. The method of operation on a brain abscess devised by Lemaitre (location of the abscess by needle puncture and drainage by a small rubber tube, increased in size at each dressing, in order to shut off the subarachnoid spaces from contamination) is highly extolled.

DENNIS, Colorado Springs, Colo.

GANGLION CELL INCLUSIONS IN THE SUBSTANTIA NIGRA. W. GODLOWSKI, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **33**:14 (Feb.) 1931.

Four cases of parkinsonism (two postencephalitic and two of paralysis agitans) were studied histopathologically. In all four the substantia nigra contained inclusions consisting of variously sized round bodies (some were smaller than a glial nucleus while others were larger than the nuclei of the large cells of the substantia nigra). Some of the ganglion cells contained only one of these bodies, while others contained two, three or four of them. With hematoxylin-iron preparations the inclusions took a rose stain, with fuchsin a very red stain and with the Mallory stain a blue color. With toluidine blue and with the fat stains they did not stain at all, nor did they take the Bielschowsky impregnation. Generally speaking, they were homogeneous and took the stains uniformly. In the Haidenhain stain, however, the center appeared like a dark granule and resembled a nucleolus. The inclusions were surrounded by a light area, which appeared to be traversed by delicate strands. In some of the ganglion cells in which these bodies were found, the nucleus as well as the Nissl bodies were still well preserved, and the cells took all stains well; others, however, were either partially or totally destroyed.

In agreement with Lewy and Redlich, Godlowski believes that inclusions in the ganglion cells in the substantia nigra are found regularly or at least very frequently in parkinsonism, whether it is due to postencephalitic states or to paralysis agitans. Their occurrence, therefore, cannot be attributed to any pathogenic factor

at the basis of either of these two diseases. It would seem rather that the ganglion cells of the substantia nigra react through their own peculiar metabolism in an identical fashion to irritants that may be entirely different in nature.

KESCHNER, New York.

OLFACTORY MENINGIOMA. C. VINCENT, H. ROGER AND MARTHE GIRAUD, Rev. d'oto-neuro-opht. **9**:571 (Oct.) 1931.

A woman, aged 47, consulted Roger in October, 1930, complaining of bilateral periorbital headache, occasional vomiting and tiring easily. Other symptoms were a tendency to somnolence, a confusional attack two months previously, defective memory, intermittent aphasia and loss of weight. Examination revealed bilateral papillary stasis, more marked in the left eye, normal retinal tension and increased pressure of the spinal fluid, which contained 0.8 Gm. of albumin and 3 leukocytes. The Wassermann test of the blood was negative. The meager findings from neurologic examinations were contradictory: a slight deviation downward of the extended right arm, increase of the right styloradial reflex, a slight pyramidal deficit in the left leg with the tests of Babinski and Barré and anosmia. A diagnosis of frontal lobe tumor at the midline, rather more on the left side, was made, and a left fronto-parietal exploration was made by Dr. Vincent; no tumor was found. At a second operation, on the right side, an olfactory meningioma, arising from the right olfactory groove and as large as an egg, was removed. The tumor occupied the midline, being more on the right side, and extended backward as far as the chiasm. Convalescence was stormy, and it was necessary to control the intracranial pressure by repeated lumbar punctures (eighteen in six weeks). Coincident with bulging of the scar, confusion of ideas, defective memory and nocturnal incontinence of urine recurred and were relieved by the punctures. The patient recovered completely. No evidence of mental abnormality remained, except that occasionally, during prolonged conversation, there was a slight dysarthria rather than a true aphasia.

DENNIS, Colorado Springs, Colo.

THE PATHOLOGIC CHEMISTRY OF THE BRAIN IN DEMENTIA PARALYTICA. M. SEREJSKY and R. TOPSTEIN, Mod. neuropsychiat. (Kharkov) **7**:52, 1931.

The brains of nine patients suffering from dementia paralytica were examined by the authors for water, cholesterol and unsaturated phosphates. The white and gray matters were examined separately and were taken in thin slices from various areas of the brain. The water content was increased slightly throughout the brain. Cholesterol was markedly increased, except in the frontal lobes. The occipital lobes had the richest cholesterol content. In the frontal lobes the cholesterol content dropped to 10.6, but in the cases in which malaria had been used in treatment, the cholesterol was increased 4.7 per cent. Unsaturated phosphates were decreased in all lobes of the brain except the occipital lobes.

The observations are consistent with the work of Pighini, with special emphasis on the definite decrease of phosphates in the frontal lobes. All this points to a lipolysis of the frontal lobes, a feature that is consistent with histologic observations. Dementia paralytica is characterized by a sclerotic atrophy of nerve cells, usually called Nissl's degeneration; there is also a lipoid degeneration. These conditions are especially definite in the frontal lobes. As the biochemical changes correspond with the histologic ones, one ought to expect that altered function of any part of the brain will coexist with a definite chemical counterpart. The patients with malarial treatment did not show the same degree of change. The author raises the question of the specificity of his observations in dementia paralytica. Similar changes were found in patients who were suffering from other neurologic conditions, such as tumors. The exhaustion from which patients with dementia paralytica usually die is another variable that may account for the biochemic observations.

KASANIN, Boston.

THE RETICULUM OF THE CILIATED CELLS OF THE LABYRINTH OF THE MOUSE, AND ITS INDEPENDENCE FROM THE NERVE ENDINGS OF THE EIGHTH PAIR. J. F. TELLO, Trav. du lab. d. recherches biol., de l'Univ. de Madrid **27**:151, 1931.

The material used consisted of mouse embryos at diverse stages of development and young mice. In addition, embryos of other mammals and of birds were examined. The ciliated cells of the cristae, maculae and cochlea contain in their cytoplasm a reticulum, which in all probability is some sort of supporting framework for the cilia. There is no continuity between the intracellular reticulum just mentioned and the nerve endings, as has been indicated by Kolmer and by London and Pesker. The nerve fibers end on the ciliated cells by means of calyces, bands or fibers enveloping the cell bodies.

The nerve fibers enter the epithelium long before the formation of the intracellular networks of the ciliated cells, but the nerve endings proper do not appear until long after the reticulum of each ciliated cell is completely developed.

As in the case of the formation of other nerve endings studied by the author, the phase of specific sensibility must be preceded by other stages of diffuse sensation, which might be compared with Head's protopathic sensibility. This sensation is probably received by the plexuses and bundles of fibers entering the epithelium before the definitive nerve endings are formed.

The cytoplasm of the ciliated cells may contain rings, first noticed by Kolmer, and seen later by Bielschowsky and Brühl. Tello regards these rings as fragments of reticulum deeply stained in partial impregnations or in altered cells. From his observations the author concludes that the ciliated cells are epithelial elements and not neurons as has been suggested by some authors.

NONIDEZ, New York.

THE HEMATO-ENCEPHALIC BARRIER IN SCHIZOPHRENIA. E. S. GILBO, J. Neuropath. & Psychiat. **1**:53, 1931.

The hemato-encephalic barrier was studied in twelve cases of catatonic, and seven of paranoid schizophrenia and in seventy-seven cases of other forms of the disease. Walter's method of the administration of bromides was used. The patients were given 10 Gm. of bromides for six days, and on the seventh day blood and cerebrospinal fluid were taken for analysis. The quantity of bromide was determined colorimetrically. The author obtained a regular curve of distribution, with a majority of the cases falling within the limits of normal, that is between 2.9 and 3.3 mg. per hundred cubic centimeters. These results are at variance with the experiments of German investigators, who found a decreased permeability of the barrier with a high permeability quotient. (This was also found in this country by Rothschild and Malamud.) The author stated that the reason that his results are different is because he had acute cases, while the other investigations concerned chronic cases.

Analyzing the material from the point of view of sex, symptoms, mode of onset and mental content, the author has found that in catatonia there is a tendency toward lowering of the permeability quotient, with increased permeability, while the reverse is true in the paranoid forms of schizophrenia. In cases in which there is a tendency toward quick recovery, there is also a lowering of the permeability quotient, with increased permeability. It is important to remember that in youth permeability tends naturally to be higher than in middle age. The same holds true for the more acute cases. In cases of chronic deteriorated forms the permeability quotient is higher, showing a decreased permeability of the brain.

KASANIN, Howard, R. I.

STERILIZATION OF THE FEEBLEMINDED. R. A. FORSTER, J. M. A. South Africa **4**:611 (Oct. 25) 1930.

According to English investigators, 80 per cent of mental defectives come from pronounced "neuropathic" stock. The neuropathic diathesis is regarded as the

cause of the minor neuroses, psychoses and epilepsy. However, this diathesis does not show itself in many descendants who are normal, but who are carriers, as it were, capable of tainting good stock. About 20 per cent of the cases of mental deficiency occur without neuropathic diathesis, and are due to prenatal, natal or postnatal injury. In 1926, it was estimated that in England 8 per cent of the population were mentally defective. Twenty years earlier the percentage estimated was only 4 per cent. In South Africa, an estimated 1.5 per cent of the population are feebleminded, and 4 per cent are possessed of only three-quarter normal intelligence. In South Africa, 1 per cent of the European stock is defective. The opponents of sterilization still hold the field, however. It has been established that the sterilization of only 17 per cent is sufficient to extinguish an entire generation. In California, the sterilization law has been in existence for twenty years, and 6,000 defectives have been sterilized, while none of the predictions of its opponents have been realized. What other means are available? There is no such thing as natural selection in civilized humanity, and isolation is too expensive. Marriage laws are a farce. Laws against copulation with imbeciles would punish about one out of a thousand. Only voluntary sterilization is legal. Sexual union is a social service only when the partners are of social value. As agreement on the principle of sterilization is very problematic, a compulsory law is the only means of handling the difficulty. As for the operation, all that is necessary is the ligation of the vas deferens and the fallopian tube.

HART, Greenwich, Conn.

AN INTIMATE ACCOUNT OF THE ORIGIN AND GROWTH OF THE MENTAL HYGIENE MOVEMENT. CLIFFORD W. BEERS, Ment. Hyg. **15**:673 (Oct.) 1931.

Beers traces his own impulse to reform conditions to a sympathetic understanding and a gift for literary expression inherited from his mother, and a persistence and hopefulness of spirit received from his father. He credits his wife with critical assistance in compiling his book, and emphasizes the encouraging effect of William James' approval of this work. He acknowledges Adolf Meyer's technical assistance and recalls that the credit of coining the phrase "mental hygiene" belongs to Dr. Meyer. He gratefully remembers the generosity of Henry Phipps and foresight of William Welch in giving and administering the National Committee's first large gift. To the psychiatric advice of William Russel and the scientific guidance of Thomas Salmon, Beers pays tribute. Many others who played important rôles in the development of the movement are cited — names like Chittenden, Stokes, Ruggles, White and Sachs. Dr. Williams is given full credit for the important work of founding and editing the journal, *Mental Hygiene*. Also recorded is the generosity of Mrs. Anderson and Mrs. Vanderbilt in financing the beginning of the International Committee. He is grateful for assistance from numerous foundations, notably the Rockefeller, the Commonwealth, the Altman and the Milbank.

The organization of the Canadian Committee by Dr. Hincks in 1917 is considered a milestone of progress as first making the movement truly international. The paper is closed with a hope and an assurance that greater financial support will be received as the movement grows.

DAVIDSON, Newark, N. J.

EFFECT OF HYPERTONIC AND HYPOTONIC SOLUTIONS ON NERVE TISSUE AND PERIVASCULAR SPACES. NATHALIE ZAND, Rev. neurol. **1**:737 (May) 1930.

Zand has repeated the experiments of Weed and of Schaltenbrand and Bailey, with the difference that after injecting distilled water into one carotid artery and 10 per cent sodium chloride into the other, she ligated the carotid and killed the animal one hour later. The alterations found differed considerably from those previously reported. In the hemisphere subjected to hypertonic solution, the nerve cells were altered in various ways; some were hydropic, others pyknotic and still others normal. Most severely altered were those of the third cortical layer. Occasional hemorrhages were visible in the superficial layers. No tissue spaces appeared around the ganglion cells. The vessels were shrunken, the perivascular spaces normal. The ependymal cells were crowded together and tended

to be detached from the underlying tissue. The endothelium of the choroid plexus was regular and rather compact; the vessels were contracted. The pia presented nothing abnormal but a few small hemorrhages. In the hemisphere into which distilled water had been injected, the tissue was furrowed, and longitudinal fissures were in evidence; also, the giant pyramidal cells were hydropic, and around them were large fissured spaces. These alterations were most notable in the cerebral cortex and in the perivascular zone. Cortical herniations were observed. The perivascular spaces were very large, and the blood vessels were dilated. The ependymal cells were stretched, flat and desquamated. The choroid plexus presented swollen epithelial cells, many vacuoles and sometimes a ruptured cell membrane. The cell nuclei were swollen and the epithelium was sometimes desquamated.

FREEMAN, Washington, D. C.

INFECTIVE NEURONITIS. HARRY G. JACOBI, Arch. Int. Med. **48**:764 (Nov.) 1931.

A young masseuse, much of whose work had been with patients suffering from poliomyelitis, was admitted to the hospital complaining of pain and weakness in the legs. Six weeks before the onset of these symptoms she had had a sore throat, from which, apparently, recovery had been complete. At first there was no objective evidence to suggest the nature of the pain and weakness, and as all laboratory and clinical tests were negative a diagnosis of hysteria was made (not by Jacobi). Within several weeks, however, she was so fatigued as to be unable to sit up. Some rigidity of the neck developed. Other signs were: lateral nystagmus, tremor of the tongue, painful areas over the muscles, paralysis of all the extremities, absent tendon reflexes, exhaustible abdominal reflexes, absent muscle and joint sense and, later, absent position sense. A fortnight later, there was a Horner's syndrome, followed shortly by dyspnea and a sense of obstruction in the throat. A terminal bronchopneumonia developed, and the patient died within seven weeks of admission to the hospital. At necropsy the neuropathologist found: a reduction in the number of ganglion cells, both in the cerebrum and the cerebellum, and a degeneration of those that were present; degenerative changes in the anterior horn cells, spinal ganglion cells, nuclei gracilis and cuneatus neurons; diffuse round cell infiltration without perivascular collars. The case is a typical one of the disease designated by Kennedy, "infective neuronitis."

DAVIDSON, Newark, N. J.

DIABETES INSIPIDUS FOLLOWING FRACTURED SKULL. A. PATRICK, Brit. M. J. **2**:689 (Oct. 17) 1931.

The case reported is that of a man, aged 21, who suffered a fracture of the base of the skull, involving the anterior and middle fossae, which was confirmed by a roentgenogram. On admission to the hospital the amount of urine passed was 45 ounces (1,275 Gm.) daily. In a week it had increased to 100 ounces (2,835 Gm.) and at the end of eighteen days to 160 ounces (4,536 Gm.). Also, at this time he was very irritable and drowsy. There were a slightly subnormal temperature, 97 F., a pulse rate of 48 and a respiratory rate of 12. Thirst was insatiable. The patient was given 1 cc. of solution of pituitary. Thirst diminished and the polyuria decreased. The total amount of urine passed in the twenty-four hours following was 59 ounces (1,672 Gm.). The temperature, pulse rate and respiration all increased. Gradually the dose of pituitary was diminished, and thirty-four days after admission to hospital he was discharged.

The symptoms in this case are explained by a lesion which is presumed to have passed through the sella turcica, in some way injuring the pituitary body, probably as a result of pressure on the pituitary gland from hemorrhage. The author reviews the literature on diabetes insipidus and shows that this condition is uncommon. His conclusions are that solution of pituitary given hypodermically is the treatment of choice, but is not a cure.

FERGUSON, Niagara Falls, N. Y.

SKELETAL MALFORMATIONS AND STRIATAL SYNDROME. CARLO ENDERLE, Arch. gen. di neurol., psichiat. e psicoanal. **12**:95 (July 31) 1931.

Enderle describes the case of a woman, aged 40, who presented: (1) radial, longitudinal ectromelia (congenital absence of the radial bone, of certain carpal and first metacarpal bones and of both phalanges of the thumb) on the right side, (2) fusion of the sixth and seventh cervical vertebrae (syndrome of Klippel-Feil), (3) anisomastia (asymmetry of breasts), and (4) torsion spasm, which developed following an abortion. Discussing various theories concerning the genesis of congenital skeletal malformation—mechanical, endocrine and cerebral—it is to the latter that he gives preference. The torsion spasm indicates a congenital lesion in the basal ganglia. Anisomastia and ectromelia may be explained as due to a congenital lesion of mesencephalic, trophic centers. The syndrome of Klippel-Feil suggests the hypothesis that the congenital disturbance in question occurred in the first two months of fetal life. A remarkable coincidence of ectromelia, anisomastia and torsion spasm in the same patient is an argument in favor of the views of De Angelis and Altschul, who believed in the existence of a mesencephalic trophic center for the mammary gland; it is also in favor of the views of Egger and Français, who believed in the existence of similar centers controlling the radial development of the upper extremities.

YAKOVLEV, Palmer, Mass.

POSTVACCINAL MYELITIS: REPORT OF A CASE. WILLIAM BROCKBANK, J. A. M. A. **97**:227 (July 25) 1931.

Brockbank calls attention to the fact that the acute inflammatory lesions of the nervous system reported as occasionally following vaccination against smallpox usually have presented the clinical symptoms of encephalitis or poliencephalitis. In cases with paramount spinal cord involvement, even when the lesions accompany smallpox itself, the sensory impairment has been negligible or transient. As a contrast to this generally accepted picture, he reports a case of postvaccinal myelitis with complete spinal anesthesia persisting up to the level of the ninth dorsal segment and paralysis. The clinical and laboratory signs of spinal meningomyelitis were presented. The sensory level seemed to indicate that the inflammation in the acute stage had progressed only to the level of the fifth dorsal segment, although motor signs pointed to mild inflammatory involvement in segments considerably higher. The sensory level two months after onset was in the eighth dorsal segment. The prodromal symptoms began on the thirteenth day after vaccination.

EXOPHTHALMIC GOITER: THE DEVELOPMENT OF REFRACTORINESS TO IODINE. WILLARD O. THOMPSON and PHEBE K. THOMPSON, Arch. Int. Med. **48**:351 (Sept.) 1931.

Sometimes a patient with hyperthyroidism shows improvement with iodine therapy, when without apparent reason the metabolic rate suddenly rises and the symptoms become aggravated. In such cases, Thompson and Thompson believe that the patient has become refractory to iodine, and that the drug should be temporarily withdrawn. After a period, varying from one to three months, iodine therapy may be resumed. The authors advise that if operative treatment is to be carried out, the operation should be performed promptly after the metabolism reaches a steady low level on iodine medication; if the metabolic rate is rising, the iodine should be withdrawn and operation postponed until refractoriness disappears.

DAVIDSON, Newark, N. J.

THE INTERVERTEBRAL DISK. EMIL S. GEIST, J. A. M. A. **96**:1676 (May 16) 1931.

Geist demonstrates in the light of Schmorl's work the anatomy, physiology and pathology of the intervertebral disk and calls attention to clinical deductions drawn therefrom in the recent literature as well as from clinical experience. He

believes that this is of importance to the orthopedic surgeon, the compensation adjuster and the roentgenologist. He discusses pathologic conditions of the disk itself, pathologic conditions of the spine in which the disk plays the preeminent part, and pathologic conditions of the spine in which the pathologic changes of the disk are of secondary nature.

MIGRAINE AND HYPOTHYROIDISM. A. LEY, J. de neurol. et de psychiat. **31**:494 (Aug.) 1931.

Ley calls attention to the association of migrainous attacks with hypothyroidism, and cites the case of a patient, aged 27, who suffered from hemicrania, the attacks being very severe, lasting about a month, and being associated with photophobia and vomiting. During the attack there were permanent vasoconstriction, loss of appetite and consequent loss of weight. There was an indefinite history of migraine in the family. Examinations of the blood and spinal fluid gave negative results. The basal metabolic rate was —16.5 per cent. Thyroid therapy gave prompt and satisfactory relief, which has continued for two years.

WAGGONER, Ann Arbor, Mich.

CASE OF PNEUMOCOCCUS (TYPE III) MENINGITIS TREATED WITH POTASSIUM PERMANGANATE—RECOVERY: A PLEA FOR ITS TRIAL. M. H. WEINBERG, J. Nerv. & Ment. Dis. **74**:38 (July) 1931.

A case of pneumococcus meningitis (type III) is described in detail. The patient was moribund on admission and recovered. The simplicity and universal availability of the treatment with potassium permanganate, which in this case was administered by rectum, 2 grains (0.13 Gm.) to 1½ pints (135 cc.), recommend it in preference to such strenuous measures as frequent lumbar punctures, cisternal puncture, ventricular lavage and injection of powerful drugs into the subarachnoid space.

HART, Greenwich, Conn.

OBSERVATIONS ON THE CALCIUM PROTEINS AND INORGANIC PHOSPHORUS IN EXPERIMENTAL VITAMIN B DEFICIENCY AND INANITION. VICTOR SCHELLING, J. Biol. Chem. **89**:575 (Dec.) 1930.

No important change in calcium, protein or inorganic phosphorus content of the blood was observed in dogs on a vitamin B-free diet, provided they took the diet. When the diet was refused and the fasting animals had lost 20 per cent of their body weight, a fall in calcium and a rise in inorganic phosphorus occurred. The convulsions observed in vitamin B deficiency are probably not due to low blood calcium.

FREMONT-SMITH, Boston.

NERVOUS SYMPTOMS AS EARLIEST MANIFESTATIONS OF CARCINOMA OF THE PANCREAS. JOSEPH C. YASKIN, J. A. M. A. **96**:1664 (May 16) 1931.

Yaskin observed depression with crying spells, anxiety, insomnia, anorexia, loss of weight and weakness without feeling of unreality and other disturbances of perception, memory and judgment in four instances of carcinoma of the pancreas in previously healthy persons. These symptoms antedated any definite gastrointestinal symptoms and objective observations by from three to eight months. No explanation is offered for the occurrence of these phenomena. The relation between visceral changes and abnormal emotional states is referred to.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

TRACY J. PUTNAM, M.D., *Secretary*

Oct. 22, 1931

A. MYERSON, M.D., *Presiding*

PARAPLEGIA ASSOCIATED WITH NONTUBERCULOUS KYPHOSCOLIOSIS; REPORT OF A CASE AND A SURVEY OF THE LITERATURE. DR. HENRY R. VIETS and DR. MILTON HENRY CLIFFORD.

It is generally considered that curvature of the spine seldom interferes mechanically with the functions of the spinal cord, and that if signs of compression of the cord develop in a patient with nontuberculous scoliosis the signs are usually due to tumor of the spinal cord, spondylitis, abscess, trauma or a superimposed disease. Paraplegia rarely develops from mechanical compression alone, due entirely to scoliosis. This fact is illustrated by a patient who entered the Massachusetts General Hospital in April, 1931, with complete paralysis of both legs. Poliomyelitis, which occurred fifteen years before entrance, when the patient was 3 years of age, had left a well marked residual kyphoscoliosis. There was no paraplegia until nine months before entry, when weakness of the legs occurred without previous trauma. Examination revealed spasticity of both legs, with loss of sensation below the level of the sixth thoracic segment. There was no sphincter disturbance and no pain. An operation was performed at the point of maximum kyphosis. The dura, which bulged posteriorly, was under extreme tension; it was opened throughout the entire length of the operative field; the spinal cord herniated through the opening; the dura was left open without drainage. Loss of sensation in the legs began to disappear within two weeks. Two months after the operation the patient was able to walk with the support of two assistants. About four months after the operation the patient reported that he was perfectly well, without numbness or weakness of the legs.

A survey of the literature shows that seventeen similar cases have been reported, the earliest being that of Mauclaire, in 1913. The most extensive work on the subject has been done by Jaroschy. Treatment has consisted of hyperextension of the spine or laminectomy, or both. All methods have been fairly satisfactory, but the patients who have been operated on appear to have made the best recoveries. One concludes that partial paraplegia may occur as the direct result of angulation of the spinal cord secondary to severe kyphoscoliosis. Paraplegia usually appears many years after the scoliosis, without predisposing trauma. The paralysis is spastic and may be associated with sensory changes and sphincter weakness. The pathway of the spinal fluid is usually blocked. With appropriate treatment, the prognosis is often good and sometimes excellent. (This article was published in full in the *New England J. Med.* **206**:55 [Jan. 14] 1932.)

DISCUSSION

DR. J. B. AYER: I remember a boy, about 17 years of age, who presented marked scoliosis with torsion. His spine had been fused by Dr. Albee years

before. There was no evidence of tuberculosis, osteomyelitis, syphilis or trauma, although it must be admitted that some pathologic condition could not be entirely ruled out. There was a partial spinal fluid block. At operation Dr. Mixter found the cord angulated by a vertebral prominence which was partially removed, seemingly relieving the tension to a great extent. There was, however, no improvement in symptoms following the operation, presumably owing to the length of time during which the paralysis had been present.

DR. T. J. PUTNAM: It is usually taught that compression of the cord cannot occur no matter how severe the scoliosis is. Did neurologic symptoms occur in any of these patients without evidence of block? When laminectomy was performed, was the spinal column weakened so that the kyphosis grew worse?

DR. H. R. VIETS: I am familiar with the case which Dr. Ayer mentions, and it should probably come in this group. In all patients examined for block, it was present to some degree. There appeared to be no further increase in scoliosis after laminectomy.

RÔLE OF OLIGODENDROGLIA IN MYELINOGENESIS. DR. L. RAYMOND MORRISON.

Since there is no sheath of Schwann in the central nervous system, the assumption is made that the oligodendroglia serve as vicarious Schwann cells as has been suggested by Hortega and Penfield. One of the functions of the cells of Schwann is obviously concerned with the laying down of myelin in the peripheral nerves. An attempt was made to ascertain whether the oligodendroglia were involved in a similar capacity in the brain and spinal cord. The "seal-ring" cells, described by Hardesty in 1905, by their appearance, occurrence and relationships in the spinal cord, suggest that they are the cells known today as oligodendroglia. Pigs were selected for these investigations, not only because of their availability, but also because they were the animals used by Hardesty in his studies on the seal-ring cells.

Embryonic pigs, from 2 to 25 cm. long, were studied by various technic, but chiefly by the Weigert-Pal, Hortega and supravital methods. It was found that myelin-staining substances first appeared in the brain tissue in the form of cells or nuclei of cells. It was observed that these cells preceded tract medullation. The study of pigs from 10 to 25 cm. proved to be most instructive. In the 10 cm. pigs, for example, there was practically no medullation of tracts and the myelin-staining cells were seen in clusters in various places throughout the brain, but chiefly around the ventricles, as has been observed by others. In the 16 cm. pig, tracts have begun to show marked myelinization, and in all cases they are surrounded by these myelin-staining cells. In the fully medullated brain there are no longer any of these cells that take the myelin stain. This suggests their involvement in the process of medullation.

Silver stains and supravital stains were used in pigs of corresponding sizes, and it was learned that many of the cells that took the stain in the Weigert-Pal preparations were oligodendroglia of youthful appearance. In fact, transitions could be followed from fat-containing embryonic cells (oligodendroblasts?) to full-blown, adult oligodendroglia; of course, with the silver stains these cells were seen to be still present in the adult brain and cord, even though the Weigert-Pal preparation did not show them to contain myelin.

One of the constituents of myelin is cholesterol-ester, which is doubly refractile; so a polarizing microscope was used to study these oligodendroglia, or their precursors, and it was found in the supravital preparations that before the oligodendroglia developed processes their perikaryon was to some extent doubly refractile, and that they contained granules that were distinctly doubly refractile and gave the typical "myelin cross."

Since there are cells that give the myelin stain and the myelin cross that precede and accompany medullation and no longer contain myelin after medullation is completed, and since these cells can be shown to be largely oligodendroglia, it is maintained that there is a relationship between the two, in fact, that oligodendroglia secrete and lay down at least some of the myelin of the nerve sheaths.

DISCUSSION

DR. T. J. PUTNAM: Dr. Morrison's work is a valuable contribution obtained through the use of a number of different forms of technic. In addition to using the classic Weigert and silver stains, he has employed a polarizing microscope for the identification of myelin and has also used supravital technic, so far as I am aware, for the first time in the study of the development of the central nervous system. From a practical point of view I believe that this work is of particular importance in relation to multiple sclerosis. The myelin appears to be laid down by embryonal oligodendroglia cells which are homologous to the sheath of Schwann cells. In the peripheral nerves, however, the adult oligodendroglia appear to be incapable of producing myelin; therefore, once the myelin is lost, as for example following encephalomyelitis, it cannot be replaced and a sclerotic plaque must result.

A CRITICISM OF CERTAIN METHODS OF CONTEMPORARY PSYCHIATRY. DR. IVES HENDRICK.

I am criticizing the overemphasis of those so-called "objective" methods of studying personal problems which deliberately exclude attention to the examiner's own emotional reaction to the subject, disregard the "psychic reality" of the subject's distortion of his experience and environment, and ignore the fact that environment after childhood is not generally a cause of personality difficulty but rather is fundamentally determined by the trends themselves. Much effort is therefore wasted in producing an illusion of "objectivity" by the accumulation of data that are etiologically and therapeutically irrelevant. Three cases illustrate this criticism: (1) A man's neurosis, character and whole development are decisively determined by reactions to an unconscious hatred and fear of a father subjectively evaluated as cruel and hostile, though objectively he is a gentle and affectionate man. (2) A woman in a hospital for mental diseases, whose behavior gives objective evidence of extreme hostility to the institution and the personnel, is subjectively motivated by a wish to deny a powerful love of these mother symbols. (3) The etiology of convulsions in a young girl is ascribed to a precocious sexual experience and "fixation" to an undesirable father; these are really superficial manifestations of intolerable jealousy reactions to profound homosexual attachments. The appraisal of these cases by what are usually termed "objective" methods produces a complete misconception. Another case is cited to illustrate how an environment apparently responsible for an acute neurosis is really its result.

"Intuition," the subjective recognition of emotional experience in another person is an indispensable tool for discovering what is concealed from objective methods, and should no longer be disparaged or regarded as mystical and unscientific guesswork. It is the "eye of psychology," the one instrument for intrinsically psychologic observation with which Nature has endowed us, and plays the same rôle in making psychologic observations as the eye in histology. But it also must be specially trained and technically controlled to become an instrument of scientific investigation. The empirical data made by its skilled utilization are then correlated, controlled, corrected and generalized by intellectual appraisal of these data. The scientific development and application of intuition is most complete in psychoanalysis, and its application to short-time psychiatric clinical work is certainly a more difficult problem. Behavioristic, environmental and interrogatory studies are essential in supplementing intuitive methods, in training the powers of psychiatric observation, and for the therapeutic amelioration of certain problems,

especially in children. I do not criticize the utilization of nonintuitive methods, but the orientation of the psychiatrist which results from overevaluation of them. Four practical suggestions are made: (1) a more systematic selection of cases suitable respectively for psychoanalysis, abbreviated subjective therapy, environmental readjustment and custodial care; (2) greater opportunities for psychoanalytic education of psychiatrists; (3) a greater utilization of social workers in psychotherapy, but with efficient supervision, and (4) less emphasis in clinical records on valueless details. One attains a maximum of objectve validity in psychiatry only by well trained subjective technic.

DISCUSSION

DR. F. ALEXANDER: This paper raises certain methodologic and philosophic questions. Recently, philosophic problems have played a great rôle in physics and in mathematics, and I do not think that psychiatry should be ashamed to indulge in them. The question of objectivity in human relations is a confusing one. The testimony of witnesses such as parents and neighbors is as subjectively involved as is the patient's testimony. Liszt, the great German jurist, caused simple scenes to be enacted before a group of witnesses who were not involved. The correspondence between the reports on the scene was extraordinarily small. The introduction of a single physical measurement into a complicated series of psychologic observations does not render the remainder of the series any more objective. This is the fallacy of the behavioristic point of view. In a certain investigation, a stop-watch was used to measure the number of minutes spent by a given child alone and when with a group in the hopes of drawing conclusions as to whether it was an introvert or an extrovert. Used without a personal subjective study of the child itself, the results are meaningless. No one would want to do away entirely with facts elicited from other people in the patient's environment, but this never gives the whole picture. Psychologic facts are more difficult to establish but no less real than facts we can see with our eyes.

DR. KARL MENNINGER, Topeka: To point out that it is impossible to eliminate the subjective in collecting data is an attack on the common sense point of view. In dealing with anything so complicated as human behavior, common sense is usually at a loss. Neurologists discovered many years ago that a person might indulge in abnormal behavior without in the least being aware of the reason. It is as if he were talking a language which common sense could not understand. One of the greatest contributions of psychoanalysis is the discovery that we cannot fully recognize and understand the subject's unconscious motives unless we understand our own. This is, however, an experience that is open to all who are willing to subject themselves to an examination of their unconscious material. We accept Dr. Morrison's conclusions about myelogenesis although we cannot confirm them except by looking into the microscope, but any one who cares to take the necessary training can also see what he sees. So with psychoanalysis, any one who cares to take the necessary training can find what Dr. Hendrick finds.

DR. J. KASANIN: I agree with Dr. Hendrick that the so-called objective point of view is a misnomer. The data acquired by social service are unsatisfactory unless verified and supported by data obtained by a subjective analysis of the patient himself. The trained intuition on which psychoanalysis rests is the most fundamental factor in eliciting really objective data. Intuition is one of the earliest forms of approach to medicine and practically the only one known to primitive healers. Unsatisfactory as it is, we have nothing at present to replace it. Possibly it may some day be replaced by some such method as that used by Pavlov in examining dogs conditioned to certain stimuli.

DR. C. M. CAMPBELL: The contrast and opposition which Dr. Hendrick has emphasized might well be replaced by the concept of supplement. Material suitable for one type of investigation may not be suitable for another. It is probably not justifiable to contrast one as objective with the other as subjective. What we are working toward are more precise methods of both kinds. We are studying

the problem of adaptation. We have learned much about the mechanisms of adaptation from physiologists, such as Cannon, from the clinical study of patients with encephalitis and from pharmacologic investigations. The anthropologist, the genetic psychologist and the child psychologist have also added to our knowledge. In the last two decades, psychoanalysis has made its contribution, although much of it may have to be revaluated before being accepted. Dr. Hendrick has emphasized one or two facts which stand out definitely. I hope that we shall be able to see that we need all these contributions.

DR. D. A. THOM: I agree with Dr. Campbell that the keynote of the whole question is cooperation. Conduct is the reaction of the individual to his environment. We therefore have to know a good deal about environment and also about the individual. We must appreciate that conduct itself is only a symptom, and that what we are concerned about is the motive behind it. In regard to the practical aspect, we have no assurance that either school is going to solve the problem for the patient, and a dogmatic point of view is not justified by either or any school as yet.

DR. A. MYERSON: By previous agreement with Dr. Hendrick, I am going to be frank. I rather object, first of all, to the limitations placed by Dr. Hendrick on what he calls psychiatry; apparently, psychiatry is only the psychoneuroses and the character problems. Nothing at all is said of dementia paralytica, pellagra, the organic brain diseases in general, dementia praecox, the manic-depressive psychoses, etc. I think we have done pretty well in the handling and understanding of dementia paralytica and pellagra. At any rate these represent two definite, objective achievements of psychiatry. The method by which Dr. Hendrick proves what he wishes to prove is simple — all too simple for a complicated world. It may be that a man's reaction to his early childhood and the complexes he formed at that date are hugely important in understanding him, but that the rest of the environment and his reaction to it on a conscious plane is of no fundamental importance will take a lot to prove. Freudian metapsychology apparently takes no account of the present moment, with its stresses and strains, its physical ill health, its economic difficulties, its ostensible marital disturbances, etc.; in other words, only a past, unearthed by a dubious technic, and which is largely discovered by symbolism, metaphor and the like, is of importance.

Furthermore, he disparages the value of the social approach made by so many American psychiatric clinics, by asserting that the freudian technic or his technic discovers the truth about the situation, which is exactly the point to be proved.

As a matter of fact, all we have to do is to turn things upside down to make a competent freudian analysis, as we see it exhibited here today. If a child likes his father, in reality he hates him; if he hates his father, he has a homosexual attachment; if he loves his mother, that is either a sign of an Oedipus complex or else he hates her, etc., *ad infinitum* and, I believe, *ad nauseam*. That man is basically a physiologic mechanism; that mood, emotion and thought are firmly grounded in the organic structure, and that fatigue is a reality do not appear in Dr. Hendrick's contribution. I think that the fact that metapsychiatry is now established as a name for the work of the psychoanalysts by their own consent sufficiently establishes the credibility of their work as science.

DR. I. HENDRICK: I feel that some confusion has been caused by my use of the words "subjective" and "objective." I chose these not because I thought that they were the best words to differentiate the two methods, but because they are the terms most commonly employed by the critics of truly psychologic study. We all understand what is meant when these inadequate expressions are used in informal discourse. Another point that apparently I did not make clear in my paper was that I was not advocating an exclusive use of subjective technic; I was merely criticizing the overemphasis of objective technic. I think that Dr. Myerson's attitude is a mistaken one in that he speaks so authoritatively about the results of a scientific method which he has never learned to use.

TRACY J. PUTNAM, M.D., *Secretary*

Three Hundred and Ninety-Fifth Meeting, Nov. 19, 1931

C. M. CAMPBELL, M.D., *Presiding*

SECTION OF THE EXTRAPYRAMIDAL TRACTS IN THE TREATMENT OF DYSTONIA MUSCULORUM DEFORMANS. DR. TRACY J. PUTNAM.

There is no accepted treatment for the distressing group of diseases known as dystonia musculorum deformans. Section of the anterior roots has been proposed, but seems almost more incapacitating than the original disease. Section of the posterior roots has been performed in athetosis, without relief. The abnormal impulses causing the spasmodic movements in dystonia appear to be discharged over the extrapyramidal motor system. In many cases, voluntary motion is little impaired between spasms. In view of the otherwise hopeless outlook, it seemed justifiable to try the effect of section of the extrapyramidal tracts.

From anatomic preparations, it can be seen that the extrapyramidal motor tracts lie in two groups. One, the rubrospinal and tectospinal, lies just anterior to the crossed pyramidal tract. The other, the so-called cerebellospinal and vestibulospinal group, lies about the periphery of the anterior quadrant of the cord. Physiologic experiments show that section of the anterolateral column leads to diminution of tone in normal and decerebrate animals. In the first two cases reported here both groups of efferent tracts were cut; in the last case only the marginal group.

CASE 1.—Harriet H., aged 14, who was referred by Dr. J. Kasanin on March 2, 1931, began to have gross abnormal spasms at the age of 6; these grew continually worse, until for the year preceding presentation she was unable to walk or to feed herself. For the month immediately preceding presentation, she was confined to bed, and the spasms, especially on the right side, became so severe that they were painful. There were difficulty in speech and emotional lability. In addition to the typical spasms, there were mild nystagmus and slight optic atrophy. The reflexes were normal between the spasms. There were no sensory changes. Irrigation of the ear-drums with cold water brought on severe contractions.

On March 26, 1931, laminectomy, with bilateral section of the tectospinal and vestibulospinal groups of fibers, was performed. The patient made a difficult operative recovery. For two weeks after the operation she was incontinent of urine and paraplegic. She had, at first, anesthesia to pain and temperature up to the waist. Occasional spasms persisted in the neck, but not in the extremities. The condition gradually improved until, at the time of discharge a month later, she was able to stand alone and to take a few steps with assistance. The hands had resumed their contracted position, but were not held as rigidly as before. There was only trifling hypesthesia to pinprick over the left leg.

On Oct. 30, 1931, the general condition had improved. The patient was able to drink from a glass placed in her hand, to write, to stand, and to walk a short distance with crutches.

CASE 2.—Thomas W., aged 12 years, who was referred by Dr. Bronson Crothers, was admitted to the hospital on June 8, 1931. At the age of 7 months, he had had a severe attack of "influenza," since which he had been in the condition shown on admission. All the extremities and the neck were the seat of almost constant tonic contraction. These contractions were made worse by excitement. On rare occasions the patient was sufficiently relaxed to pick up an object from the table or floor. However, he was never able to stand, and had great difficulty even in sitting upright. When in the presence of strangers he was constantly rigid and usually bathed with perspiration. Owing to irregular respiration and phonation speech was difficult, but his mentality was excellent. In the intervals between the spasms the reflexes were normal, and sensory examination gave normal results.

On June 29, 1931, laminectomy was performed, with section of the rubrospinal and vestibulospinal groups on the right at the level of the fourth cervical and on the left at the fifth cervical nerve. The patient made a fairly good recovery. He was incontinent for a few days, and had a severe headache. He was able to move all the extremities, though feebly at first. There were no sensory disturbances at any time. Spasms recurred in the left trapezius and the levator scapulae group, so that the arm was flexed and raised. There was torticollis, and speech was unimproved. Only very mild spasms occurred elsewhere in the extremities, and the patient gradually recovered partial use of the limbs. A bilateral Babinski sign persisted. When the boy was last seen on Oct. 14, 1931, the spasms in the left shoulder persisted. He was unable to stand, but could sit in a chair comfortably. There was weakness of the neck which made it necessary for him to wear a supporting collar.

CASE 3.—Beverly H., a sister of Harriet H. (case 1), who was also referred by Dr. Kasanin, was admitted to the hospital on Nov. 11, 1931. The symptoms were similar to those of the sister, but begun at the age of 5 years and had grown worse more rapidly than those in case 1. She had almost constant spasms of the right side of the body, especially of the arm, which was practically useless. She could not stand, and sitting was difficult. She could help herself somewhat with the left hand and could write with it, but she could not hold a block of paper.

On November 13, bilateral section was made of the anterior marginal group of tracts, on the right at the level of the third cervical nerve and on the left at the fourth cervical nerve. The patient made an excellent operative recovery, was not incontinent, and had no sensory loss. Three days after operation, there was a left Babinski sign. Feeble movements were possible at once in all the extremities. The fingers, which had been tightly clenched, could be extended for the first time in several years. All that remainded of the spasms was an upward twitch of the right shoulder.

At the time of presentation, three weeks after the operation, the twitching of the right shoulder continued, but was less constant as the patient became stronger. The deep reflexes had reappeared in all the extremities. The Babinski sign was absent. The legs could be moved freely and with fair strength, though they were not yet strong enough to support the patient's weight. The left hand had become clenched, though less firmly than before the operation. The right hand could be opened and closed at will, though weakly. The patient's general condition was much more tolerable.

DISCUSSION

DR. J. KASANIN: Dr. Putnam's first patient is immeasurably better than before operation. The grotesque athetoid movements have disappeared, except to a slight degree in the upper extremities. Her personality is much improved, and she is easier to live with. I believe that with exercise she could walk much better, but she is not making any serious effort.

DR. E. W. TAYLOR: This work seems to me to be very important and to offer many possibilities for the future when the technic is perfected. Dr. Putnam is wise in not sharply distinguishing types but regarding all the forms of the condition as belonging to the group of lenticular degeneration. I should suppose that the operative procedure that he has described might also be applied to various athetoid conditions. The point of chief importance, however, is that the extrapyramidal tracts have been isolated and can be operated on without undue damage to adjacent structures. Even a little relief is obviously of great value even though no cure is accomplished. Possibly something may also be done by retraining. In a young patient with dystonia musculorum deformans observed at the Massachusetts General Hospital there were continual violent athetoid movements so that he was apparently incapable of any purposeful activity. When, however, a ball was thrown to him in play all movements at once ceased and he caught the ball with a high degree of coordination. Naturally, such sessation of irregular movement is temporary but, as occurs in Parkinson's disease, it seems possible that a certain degree of coordinative movement of a voluntary sort might ultimately be attained.

DR. H. C. SOLOMON: Would it be possible to cut the tracts higher and more posteriorly in the medulla, or perhaps even to section the auditory nerve, which of course would leave the patient with permanent deafness. Much can be done by reeducation in such cases. A few years ago, in New Haven, I saw a man who was at that time a student in a medical school. He had had dystonia musculorum at the age of 12 in such a severe form that he was unable to talk. He had made up his mind to control the condition and had succeeded in doing so.

DR. C. M. CAMPBELL: Has this operation been performed in any other conditions of this group, such as torticollis, chorea and Parkinson's disease? Dr. Taylor's case in which the movements ceased during play points the way, I think, to reeducation. The symptoms might be controlled by keeping the patients keyed up to the effort required in play.

DR. T. J. PUTNAM: My associates and I have, of course, thought of other diseases in this group and I have at present in the hospital a patient with severe encephalitic paralysis agitans who has not been benefited by section of the tracts. For torticollis, one would have to section at a much higher level, but this might be done. I am going one segment higher with each operation that I do. The technic is easier and safer than that of cutting the vestibular nerves. Remarkable results have been secured by retraining both by Dr. Crothers in this city and by Dr. Phelps in New Haven. The second patient whose case I reported here had had a long course of treatment at the Children's Hospital with little benefit, and the other two patients had been examined there. I think that it is possible that partial control of the abnormal movements by section of the tracts would make reeducation easier. It is difficult to test coordination after these operations, but there is certainly relatively little loss. The first patient has regained ability to write and the last one can do the heel-to-shin test and knuckle-to-nose test in spite of section of the spinocerebellar tracts.

ACTION CURRENTS IN THE CENTRAL NERVOUS SYSTEM: I. ACTION CURRENTS OF THE AUDITORY TRACTS. DR. LEON J. SAUL and DR. HALLOWELL DAVIS.

This paper will be published in full in a later issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.

PAVLOV'S THEORY OF SCHIZOPHRENIA. DR. J. KASANIN.

This paper is a translation of what seemed to me the most important passages in two articles read by Pavlov before the Postgraduate Medical Assembly of Leningrad on Jan. 12, 1930. The first was entitled, "The Physiology and Pathology of the Highest Nervous Activity," and the second, "An Experimental Excursion of a Physiologist into the Field of Psychiatry." Both of these papers were published in a small booklet by the State Medical Publishing Company of Leningrad and Moscow. The translation of the selected passages follows.

"Studying the highest nervous activity of dogs, we found a certain definite type of response of the central nervous system. A study of a large number of dogs, conducted for over thirty years, led to the development of a classification closely allied to the old hippocratic theory of temperaments. In the first place we found two clearcut types, the impetuous or excitable type, and the inhibited type. I have already pointed out that all conscious activity of animals consists of an interaction of two fundamental processes, the process of excitation and the process of inhibition. In our dogs we found, on the one hand, the impetuous type, characterized largely by easy excitability and lack of strong inhibitions and, on the other, an inhibitory type with a well expressed dominance of inhibitory processes and quick exhaustion of excitations.

"Let an example of the impetuous type be taken. A positively conditioned food reflex is formed to a certain rate of tone vibrations, and then a negative reflex is established to the next frequency of vibrations in which no food is given.

The dog of the impetuous type has difficulty in controlling responses. In spite of repeated training, he still responds to the negatively conditioned reflex with a slight amount of salivation, although no food is given. Consequently, the dog of the impetuous type lacks inhibitions, and the process of excitation is stronger than the process of inhibition. At the same time, this dog can stand a great number of stimuli without showing any ill effect. The inhibitory type of nervous system presents a different picture. A dog of this type quickly forms conditioned inhibitions, but shows inadequacy of its responses to excitation. One no sooner applies a strong stimulus than the dog becomes more or less inhibited and completely incapable of nervous activity.

"Between these two extreme types is found the medial type, with well balanced excitations and inhibitions. In our material the well balanced type can be differentiated into two subtypes, one that is naturally calm and collected while the other is more alert and vivacious. If we apply to these types the ancient classification of temperaments, then the impetuous type would be called the choleric and the inhibited type would be grouped with the melancholic; the well balanced type would correspond to the quiet or phlegmatic type or, on the other hand, to the sanguine type, i. e., a subject that constantly demands stimulation for his activity.

"With this introduction on the physiologic study of the central nervous system, I can pass to the more pathologic aspects of our material. As one might guess in the beginning, the deviations from the norm represented by the two extreme types are the pathologic or unbalanced types. You know that normal nerve activity is a result of two processes, excitation and inhibition, in a very complex interrelationship, and you can well imagine that the extreme types, which are incapable of integrating and coordinating their fundamental nervous processes, will naturally break down much sooner under various stresses and strains.

"This conclusion is supported by experimental facts. If a dog of the impetuous type has to balance the processes of inhibition and excitation under difficult conditions, as, for example, when the conditioned inhibitory stimulus follows immediately after an excitatory stimulus, the animal becomes extremely excited and loses its capacity for the development of inhibitions. The state of marked irritability of the nervous system as a result of close juxtaposition of the excitatory and inhibitory processes with a loss of capacity for inhibitions I have called neurasthenia, which of course, is incorrect philologically.

"To what extent this condition compares with the clinical concept of neurasthenia is beyond my competence, and can be decided only by a neuropsychiatrist. We have had cases of such neurasthenia in our laboratory. We were able to produce the condition experimentally and, what is more important, we even treated the animals and restored mental health. In this respect we were helped by medications that are used for human beings, especially bromides. Apparently, if bromides are used occasionally for several weeks, the animal regains a capacity for solving difficult problems.

"Turning to a study of the behavior of inhibited animals under similar conditions, if such an animal is subjected to very strong stimuli and the inhibitory and excitatory stimuli follow one after the other without any interval, the animal becomes unable to respond to more conditional stimuli. In my cases we noticed that the dogs lapsed into a chronic inhibitory state, even losing their capacity for responding to weak stimuli. A remarkable illustration of this phenomenon was supplied by the great flood at Leningrad in 1924. The dogs in our laboratory were in danger of drowning. Through the general tumult of the flood, the roar of falling trees and high waves attacking the building, the dogs had to be rescued by making them swim a certain distance and then getting them to the upper stories of the building. As a result, many of the animals became seriously ill. The strong, impetuous dogs stood the test very well, while the inhibitory dogs became sick. They lost all the reflexes that they had learned and had to be helped even with the conditioned food reflexes, i. e., they had to be coaxed to eat. Some of the dogs never recovered from this nervous state. There is an

interesting case of a dog that becomes incapacitated whenever we apply strong stimuli. When many stimuli are applied at the same time he becomes completely inhibited. This dog can work only when he is in his fixed, favorable environment. Such a state, in which the nervous activity under the influence of strong stimuli expresses itself largely in inhibitions, I have named hysteria, qualifying this term, of course, in relation to dogs.

"It is curious to note that the objection to the use for dogs of the definition of hysteria in human beings is based on the fact that in human beings in hysteria there are not only states of inhibition but marked states of excitement. This objection, however, does not contradict the experimental facts. The dogs of inhibitory type often show outbursts of excitement. I may cite the example of a dog of this type that had to be kept in a holder for long periods. This docile and rather cowardly animal would become extremely excited after being released from the holder. I shall bring up later some facts explaining this phenomenon.

"There is another fact that constantly comes up in our material and that can perhaps explain some of the pathologic processes of the central nervous system. It has already been pointed out that the brain is a complicated organization, all parts of which interact with each other. However, one can isolate one area by incapacitating it while the activity of the rest of the brain remains unimpaired. Suppose that a series of conditioned reflexes were established, various sound stimuli, such as noises, ringing of bells and metronome vibrations, being used. Each one of these stimuli acts on some definite area in the cortex. It is possible to put out of order any point in an analyzing area of the cortex without disturbing other points in the same area. Suppose that an animal learns to differentiate between two frequencies of tone vibrations. It forms a conditioned food reflex to a vibration rate of 100 beats per minute, and a negative reflex not supported by any food to a frequency of 95 vibrations per minute. Thus an inhibitory reaction to 95 vibrations per minute develops. After these conditioned positive and negative responses have been established, one begins to apply the stimuli one after the other without any interval, occasionally reversing the order. An overlapping of the positive and negative stimuli is produced, and a specific analyzing area in the cortex becomes incapacitated or sick. The law that states that the response is directly proportional to the intensity of the stimulus does not hold true. To weaker stimuli one secures much stronger responses. The cells in the analyzing area are weakened and cannot stand strong stimuli. If the sound analyzing area is still more deeply involved, even weak stimuli give no response. Both weak and strong stimuli result in complete inhibition at the given point which spreads all over the cortex, completely modifying the behavior of the animal. At the same time, all other points in the sound-analyzing area of the cortex remain unimpaired; thus, if other sound stimuli are applied the response and the general behavior are normal; as soon as the metronome begins to swing at the rate of 95 or 100 beats per minute the paradoxical phase of inhibition develops, and all conditioned reflexes disappear; this state may last for several days.

"A study of the hypnotic state of our animals led to an understanding of some symptoms encountered in mental diseases. As already stated, between the two extremes of the waking condition and sleep there are transitory states which appear in dogs as phases of gradually increasing inhibition. Inhition developing in one area of the cortex spreads partially through the rest of the cortex, attacking some areas and leaving others free, varying not only in the direction of the spread but also in the intensity of the process. As an illustration, a conditioned food reflex usually expresses itself in two ways. On the one hand, there is increased salivation, and on the other hand there occurs the motor reaction to food—the dog looks at the food, tries to reach it, opens its mouth, etc. But as soon as the animal lapses into the hypnotic state, no matter what the cause, there appears evidence of dissociation. On application of the conditioned reflex the saliva begins to flow; consequently, the stimulus is acting; but when the dog is given food he refuses it. A strange picture! The animal becomes stimulated by the signal, and yet does not take the food. The explanation is simple. As soon as the hypnotic

state is broken, the animal begins to eat. This striking fact has a simple explanation: Voluntary movements originate in special areas of the motor cortex, and although other areas of the cortex escape, including those that control salivation, the motor areas are inhibited.

"This is an example of dissociation of the activity of the cerebral hemispheres. The motor areas of the hemispheres are inhibited, while other areas remain active. Another detail in the experiment makes it particularly curious. It appears that the whole motor area is not inhibited at once, but that the inhibition progresses in a certain order which has a firm physiologic foundation. When the dog is given food, a series of kinetic phenomena appear. The trunk turns in the direction of the food, the cervical muscles flex the head, the muscles of mastication begin to contract, etc. Inhibition as well as excitation follows a certain order. When the hypnotic state begins, the first sign that the dog shows is inability to contract the muscles of mastication, other motions being left free. There appears the following picture: The conditioned stimulus is applied, the saliva begins to flow, the dog turns to the food and bends its head, and yet cannot take the food, the tongue protrudes as if it were paralyzed and the animal is unable to open its mouth and chew. This process coincides with other facts in pathology—the inhibition, or the pathologic process, attacks first those areas which previously were most active.

"Other states follow. The dog will turn its body but will not bend its head; if the inhibition spreads, even the turning of the body disappears and the dog remains standing in one place. When inhibitions spread through the whole hemisphere, the dog becomes cataleptic; it stands as if ossified, all motions disappear, and no reflexes can be elicited.

"It is obvious that the inhibition not only spreads through the hemispheres but attacks the basal ganglia, without disturbing, however, certain centers dealing with the sense of motion and position. When, however, inhibitions spread even to these centers complete sleep with relaxation of the musculature occurs.

"Thus, inhibition may be differentiated according to its intensity and localization; dissociation of the activity of the brain takes place not only in the cortex but in the subcortical centers, giving rise to a variety of phenomena. It is easy to imagine how rich this variety is in man, who differs from other mammals in the size of his brain and in the tremendous complexity of its functions. The general principles of higher nerve activity are the same in man as in the higher mammals. I was convinced of this after I saw the behavior of patients in the wards. Due to the courtesy of Professor Ostankov and his associates I saw cases of the various forms of dementia praecox. Comparing them with my own material, I came to the conclusion that the various stages of the disease are merely phases of a hypnotic state, which can be explained in the light of our experimental data which deal extensively with the process of inhibition. In order to make this conclusion clearer I shall discuss an extremely important feature of our work, namely, the relationship of the cortex to the subcortical areas.

"The higher nerve activity is composed of the activity of the cortex and of the basal ganglia, representing a united activity of these two important parts of the central nervous system.

"The basal ganglia are the centers of the most important unconditioned reflexes or instincts, such as those of food, self-defense, sex, etc., representing the main life stream of the living organism. In the subcortical centers are the foundations of overt activities of the organism. From a physiologic point of view the subcortical centers are characterized by their inertness to stimuli, whether excitatory or inhibitory. The dog in which the cerebral hemispheres have been removed responds to only a few of a great many stimuli to which it is exposed. The outside world is thus narrowed. The same dog is unable to extinguish its reflex response. For example, it is incapable of inhibiting its orienting reflex to frequently repeated stimuli, while, in a normal dog, the reflex becomes extinguished after it is repeated four or five times. The functions in the hemisphere as compared with the basal ganglia consist in a fine, extensive and delicate analysis of

all stimuli coming from within or without. Compared with the crude activity of the basal ganglia, the cortex is responsible for the finer movements of coordinating the organism more precisely and accurately with its environment. On the other hand, the basal ganglia are the source of crude activities which are eventually utilized by the cortex. Everyday facts demonstrate the influence of the subcortical centers on the cortex. A hungry animal that has not been fed for twenty-four hours will show large conditioned food reflexes, but a dog recently fed will give much smaller responses.

"I have pointed out that the amplitude of a response depends on the strength of the stimulus, provided there is a constant food intake for twenty-four hours; but as soon as responsiveness to the food is stimulated, either by decrease in the ration of food or by delay of the meal, the rule no longer holds true. Both weak and strong stimuli elicit the same degree of response. What appears more often is that weak stimuli produce stronger responses than strong stimuli. This is what I call an equalizing or paradoxical phenomenon. By reversal of the experiment, i. e., decreasing the responsiveness to food in the dog by feeding him just before the experiment, the same response is obtained to both kinds of stimuli. There is, however, a fundamental difference. The equalization of responses takes place on a higher level in the first series of experiments than in the second series. Eventually, the dog does not take food with strong stimuli but does take some when weak stimuli are applied. In both cases the strong stimuli lose their potency, whether the dog is hungry or whether he has eaten just before the experiment; the degree of response falls below the usual norm. This can be explained by the fact that when sensitivity to food is raised, strongly stimulated subcortical centers charge the cortex, increasing the lability of the cells. The strong stimuli become supermaximal, eliciting an inhibitory reaction. Conversely, in lowering of the responsiveness to food, the impulses from the basal ganglia are weak and the lability of the cortical cells becomes decreased, especially of those cells that were under stronger stimulation previously. The influence of the subcortical centers on the cortex is seen clearly when only weak stimuli are applied. A definite law becomes apparent—the effect of the weak stimuli depends directly on the lowering or heightening of the responsiveness of the animal to food, i. e., it becomes increased with heightening of the sensitivity to food and decreases with its lowering.

"This influence of subcortical centers on the cortex is substantiated by other experiments. A dog with fatigued cortical cells, in which the reflexes were very weak, was subjected to ligation of the vas deferens and transplantation of testes from another dog. This was probably followed by an increase in secretion of the sex hormone. The operation was beneficial to the dog, all reflexes were restored, and the nerve cells again became capable of solving difficult problems. The beneficial results lasted only a short time. Within two or three months, the dog returned to the original state. When gonads were removed from normal healthy dogs, a marked disturbance of the nervous system developed, which resembled clinically an attack of hysteria or perhaps early dementia praecox.

"The subcortical centers are a source of energy of all higher nerve activity, and the cortex becomes the regulator of this blind force, controlling it and directing it. The inhibitory influence of the cortex, already established by Setchenoff, becomes apparent in one of our experiments which may be of importance clinically. I saw an ordinary case of war neurosis. The patient, a former colonel in the army, was well during the day, but whenever he fell asleep he became very excited, waved his hands, yelled and issued commands; in other words, he lived through his experiences in the war every night. We were able to reproduce a similar condition in a dog. A dog was trained to respond to various sounds of the same musical instrument in connection with some other unconditioned reflex. One of the reflexes was connected with injection of acid, others with reactions to food, and still others with very strong stimulation of the paws with an electrical current. The electrical stimulus applied was so strong that a very strong defense reaction developed, with an attempt to break away from the holder, loud barking,

etc. The defense reaction was so intense that the dog responded in the same way to the giving of food and acid, even though the two types of stimuli were never applied together. After a while the experimentation was continued with only the food reflex, while the acid and the electrical stimulus were dropped. The defense reaction in connection with the food reflex also gradually disappeared within two months. Still later another fact was observed. When the dog was hypnotized, as was shown by the paradoxical reflex, he would show a marked defense reaction immediately after eating. As soon as the hypnotic state was lifted, the defense mechanism disappeared. The analogy with the clinical case was striking. In both cases the severe experiences took place a long time before; yet their dynamic influence revealed itself after the subjects lapsed into sleep. This can be explained by the theory that the subcortical centers retained traces of former strong stimuli, which became apparent only when the cortex was inhibited or when there was a positive induction from the cortex into the subcortical centers.

"After one has become acquainted with the activity of the cortex and of the basal ganglia in general, my interpretation of schizophrenia as an inhibitory state of the cortex becomes clear. My attention was called to a symptom which apparently has no special name in psychiatry. The symptom consists in the fact that the patient does not reply to a given question and refuses contact with the examiner; yet the same patient may talk to the physician when he is put into quiet and pleasant surroundings. This is the same situation as that of the dog which does not respond to strong stimuli but does respond to weak ones. I understand such symptoms as echolalia, echopraxia and stereotypy as various stages of a hypnosis, which concentrates its action in one or more parts of the cortex. There is sufficient evidence to warrant looking on schizophrenia as an inhibitory state of the cortex protecting the nerve cells from further exhaustion. The mild facetiousness and silliness in hebephrenia can be explained by an attempt of the subcortical centers to free themselves from the inhibitory influence of the cortex.

"I have called attention to the great variability of the hypnotic states in animals and to the dissociation in the activity of the hemispheres when some areas in the cortex are stimulated while other areas are inhibited. It is easy to imagine how great this variety is in human beings. It would require a tremendous effort to orient oneself in this complexity, but I feel some satisfaction in our work, not simply because some of the facts that we have discovered can be applied to man, but because we have developed a method for getting facts that will help in the study of the highest nerve activity.

"In our material, we have produced cases of neurosis experimentally in dogs, and we believe that it is possible to produce in dogs other conditions that would resemble psychoses in human beings. This was why I decided again to study psychiatry, which I had forgotten after my student days. Owing to the courtesy of my colleagues, Professors Ostankov and Narbutovich, I had an opportunity to observe the various psychoses. The first group I studied was schizophrenia. Here my attention was called on the one hand to the symptoms of apathy, dulness, stupor and stereotypy, and on the other hand to the facetiousness, silliness and, in general, childish behavior, which the patients had not shown before the illness. What are these symptoms from a physiologic point of view? Can they be understood and thought of in physiologic terms? For this one must turn to the data that we have obtained in our work with conditioned reflexes. These data throw a good deal of light on the process of inhibition in its physiologic and pathologic aspects.

"Inhibition, taking an equal part with excitation in the everyday activity of the animal, also acts as a guardian of the most active cells in the brain, i. e., the cells in the cortex, protecting them from too much exertion when they are stimulated by very strong stimuli or when they are continually stimulated by weaker stimuli. It is the function of inhibition, then, to secure for the cortical cells a period of normal rest after a long period of activity.

"I am certain that sleep is nothing else than an inhibition that has spread over the cortex and has penetrated to a certain depth into the brain. We have also had

the opportunity to study in our animals the intermediary stages between sleep and wakefulness, that is, the hypnotic phases. We think of these phases, on the one hand, as various degrees of intensity of inhibition, i. e., greater or lesser diffusion of inhibition in various areas of the brain; on the other hand, we look on them as degrees of intensity of inhibition of varying depth in the same area of the brain. With the tremendous complexity of the human brain, the variety of hypnotic states in man is great. But some hypnotic states are more pronounced in animals than they are in man; even in man the hypnotic states show a great deal of variability, depending on the individual patient and on the method of hypnosis. For this reason, realizing the complexity of the subject, I shall consider the hypnotic states both in animals and in man.

"A study of the schizophrenic symptoms mentioned led me to the conclusion that they are an expression of a chronic hypnotic state, a point of view that I shall maintain in this paper. True, apathy, dulness and immobility are no proof of the hypnotic states, but neither do they contradict this view, especially if my thesis finds confirmation in other more specific symptoms.

"First, I shall cite the following facts: Usually the patient is considered apathetic and dull because he does not reply to the psychiatrist's questions. It is considered that his emotional life is impaired. But if one asks the same questions quietly in a pleasant informal way, one often gets a reply. This is a striking phenomenon to which little attention is paid. In my opinion this symptom is one of the most definite evidences of a beginning hypnosis. We constantly see this paradoxical phase when strong stimuli elicit no response but weak stimuli give a normal reaction. In a case of hypnosis of five years' duration described by Janet, this was the way in which he was able to establish a contact with the patient. The patient was freed from her hypnotic state when all strong stimuli were removed. Then, in schizophrenia there occurs what is commonly described as negativism. In our experimental animals, negativism is commonly encountered in the beginning of the hypnosis. With the conditioned excitation associated with the food reflex, the dog is offered food, but stubbornly refuses it. One detail is signficant. When the food box is removed the dog tries to reach it. This may happen several times in succession. No sooner is the hypnosis broken than the dog immediately laps up the contents of the food box which he had previously refused.

"Another symptom of schizophrenia is stereotypy, i. e., a tendency toward repetition of various movements, postures, attitudes, etc. This is also a hypnotic phenomenon. In some of our dogs it can be observed clearly. A dog fully awake, after he gets through eating in a setting of a conditioned food reflex, often begins to lick his own paws and chest and the anterior part of his body. In the beginning hypnosis this licking becomes extremely prolonged, going on hour after hour until the next feeding. Some other motions also tend to be repeated indefinitely.

"A frequent symptom of schizophrenia is catalepsy, retention of certain fixed postures with the accompanying phenomenon of waxy flexibility. A more dramatic symptom is catatonia, a state of marked tension of the skeletal muscles that resists attempts at relaxation coming from the outside. Catatonia is nothing but a discharge of tonic reflexes, such as occurs in the subject who becomes rigid as a board in hypnosis.

"The variety of symptoms induced by central inhibition is enriched by the playfulness and silliness seen in hebephrenic subjects, as well as the outbursts of excitement with aggressiveness that are observed in other groups of persons with schizophrenia. These symptoms resemble the picture of beginning alcoholic intoxication or the behavior of young puppies or infants on awakening or falling asleep.

"All these symptoms can be looked on as a result of beginning inhibition of the cortex, so that the subcortical areas not only are released from the constant inhibition exercised on them by the cortex but get into a state of chaotic excitation by virtue of the mechanism of positive induction. Hence, in alcoholic intoxication there are happiness and hilarity without rime or reason or excessive sentimentality with crying or outbursts of anger. The same phenomenon is seen in

children with tantrums of temper before they fall asleep. An infant, aged 6 months, when falling asleep presents a remarkable picture. In the baby's face there is a kaleidoscopic variety of expressions due to the chaotic activity of the paleo-encephalon. In the various stages of the disease a schizophrenic patient presents the same phenomena, either in long spells or as temporary explosions.

"After all that has been said, it is apparent that schizophrenia in some of its phases and manifestations is a state of chronic hypnosis. The state may last for years. Bearing in mind the case of somnambulism, of five years' duration, reported by Janet, and a case reported in St. Petersburg which lasted twenty years, why cannot hypnosis last for years, especially when the condition in the cases cited resembles hypnosis more than sleep?

"What causes the chronic hypnosis of schizophrenia? What factors are physiologic and which are pathologic? What are the cause and the outcome?

"The fundamental cause of the hypnosis is a weak nervous system, with special weakness of the cortical cells. This weakness has many causes, both constitutional and environmental. I shall not discuss these causes here, but it is obvious that such a nervous system becomes quickly exhausted when subjected to various physiologic stresses as well as to unfavorable environmental factors. Exhaustion is the strongest physiologic stimulus for the appearance of inhibition as a protective reaction. Hence, there appears a chronic hypnosis, an inhibition in its various manifestations. This state is pathologic, because it robs the patient of his normal activity, but by virtue of its mechanism it is really physiologic. It protects the cortical cells from the complete destruction with which they were threatened by the impossible load. In our laboratory we have an example of prolonged inhibition which restored to the weak cortical cells the capacity for normal activity. There is reason to believe that while the inhibitory process is acting the cortical cells are no longer damaged and that there is a chance for a return to normal function—that the pathologic reaction is reversible. This process, in accordance with modern terminology, means a functional disorder. That this is so can be confirmed by the following fact: The catatonic form of schizophrenia, that is, that form of the disease that is characterized particularly by hypnotic manifestations, shows a fairly high percentage of recoveries, which does not hold true in other forms of the disease.

"In conclusion, permit me to make a therapeutic suggestion, even though it may sound sentimental. No matter how magnificent has been the progress in the treatment of the mentally ill, there is something lacking. The mixing together of patients with various degrees of insight and behavior and the lack of segregation of quiet from extremely noisy and excited ones who undoubtedly annoy the quiet patients and sometimes abuse them can be looked on as an additional load on the already weakened cortical cells. The realization of the loss of freedom, which, of course, may be necessary, also 'adds insult to injury.' It seems to me that mentally sick patients should be treated, as are all other persons who suffer from physical disease, in such a manner that the treatment does not rob the patients of dignity and self-respect."

DISCUSSION

DR. F. ALEXANDER: The tendency to explain psychologic facts by physiologic facts is as old as the knowledge of the central nervous system. Of course, steady progress has been made. In the seventeenth century Descartes considered the pineal gland to be the seat of the soul because it was in the center of the brain. Later materialistic philosophy considered thoughts glandular products of the brain. In modern times such investigators as Vogt have done much to distinguish between the relative rôles of the cortex and of subcortical centers in such relatively simple psychologic processes as pain. The psychoanalyst's approach seeks to explain the seemingly irrational behavior of insane persons by similar correlation of psychologic facts. My chief objection to Pavlov's theory is that it falls back into old mythology, explaining facts by a vague generalization such as a hypnotic state. Breuer, sixteen years ago, compared schizophrenic thinking to dream thinking. Breuer went further than Pavlov, however, and demonstrated that the patient acts

out his dream phantasy. Pavlov candidly remarks that he does not know much about psychology. He disregards all the psychologic details of schizophrenia and tries to reduce their complexities to a few physiologic generalities. This can be done only with great inconsistency. Pavlov's pupil, Schinson, has published two books on the subject which have a great advantage over Pavlov's discussion because Schinson is familiar with both physiology and psychology. He did not try to go so far as to explain schizophrenia. I should like to mention only one detail which illustrates all possibilities of cooperation between physiology and psychology in psychiatry. This is the fundamental fact of repression which appears to be really a conditioned inhibition. Many of our patients show inhibitions to a certain type of woman, that is, blond or brunette or to figures belonging to a certain sex group. Some men have a conditioned inhibition to women of the same class as themselves. Psychologic research shows that these negative conditions or characteristics are the same as those of the first object of sex longing, usually a female member of the patient's family. The repression may be so great that the inhibition radiates to all women, and the patient may become entirely confused and inhibited. This is similar to the condition in the dog stimulated by electrical current at the same time that food is given. I hope that I have succeeded in showing you that such kinds of comparison are much more useful than Pavlov's.

DR. I. CORIAT: Pavlov has interpreted the total personality of the schizophrenic person on the physical level, ignoring the deeper mental mechanisms.

DR. H. DAVIS: It seems to me that there is an inflation of definition in Pavlov's interpretation of his work which has reduced the predictive value of his conclusions.

DR. J. KASANIN: It is not my intention to defend Pavlov's thesis but merely to present it and allow it to stand on its own merits. I think that the defect in his theory lies rather in the things he does not explain than in any errors in explanation. For example, he does not say anything about hallucinations or feelings of hostility or about the phantastic and symbolic misinterpretations of the environment. On the other hand, he gives excellent psychobiologic explanations of some of the most cardinal symptoms of schizophrenia, such as withdrawal and stupor. Pavlov paves the way toward the introduction into the field of schizophrenia of the experimental method, without which no real research is possible.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 27, 1931

JOSEPH MCIVER, M.D., *President, in the Chair*

TRANSIENT HEMIPLEGIA IN A SYPHILITIC PATIENT OF TWENTY-EIGHT. DR. MELBOURNE J. COOPER.

The interesting features in this case were the occurrence of a complete right hemiplegia, accompanied by a disturbance of speech, in a syphilitic patient, aged 28, the symptoms subsiding to some extent after an hour, and essentially normal function returning within two months after the acute attack.

The patient, a woman, gave the history of a "positive" blood test three years prior to presentation. At that time she had submitted to three treatments with injections, given in the arm, but she had not completed the course of treatment advised. About Sept. 15, 1931, she experienced an attack of vertigo which was followed immediately by subjective and probably objective sensory loss in the extremities of the right side, and a few moments later by a complete loss of the power of voluntary movement of the right upper and lower extremities. There were a coincident complete loss of the power of speech and a feeling of faintness and

nausea, but no loss of consciousness. She was unable to say a single word at the time, and kicking the left foot against the right caused no sensation in the latter. An hour later, she began to regain a little power of movement of the affected extremities and was able to utter a few words, but speech was indistinct. Five or six hours later, she could walk a little and speech was a little more distinct. About two days after this, she walked four city squares, but felt very tired and weak after the effort.

The patient was seen in the service of Dr. William G. Spiller at the neurologic outpatient department of the University Hospital on Sept. 25, 1931. Dr. Cadwalader's examination on that date showed that there was a residual weakness of the right side, including the right side of the face. The patient stated that there was some difficulty in swallowing solid food, and that this difficulty had been present since the acute attack. The voice was husky, but the movements of the palate appeared normal. Dr. Houser found no paresis of the vocal cords or soft palate, and a fluoroscopic examination to determine the function of swallowing showed no impairment. The pupils were equal and normal in size and reacted normally, and the ocular rotations were normal.

On September 28, further examination showed that the tendon reflexes in the right extremities were considerably increased, and the right abdominal reflexes were absent. The tendon reflexes on the left side were moderately hyperactive, but less so than on the right; the left epigastric reflex was lost, and the left lower abdominal reflex was preserved. There was no Hoffmann or Babinski sign on either side, and no clonus. The weakness of the right side of the face was of the upper motor neuron type. The pronunciation of words used in ordinary conversation was indistinct at times, and there was decided difficulty in pronouncing many syllables of the usual test phrases. No aphasia of any form could be demonstrated. There was no deviation of the tongue, and its movements in all directions were free. The fundus and the peripheral visual fields showed no abnormalities. The Wassermann reaction of the blood was strongly positive. A general physical examination revealed no essential abnormalities; the heart was normal, and the blood pressure was 110 systolic and 60 diastolic.

On October 3, the patient had an attack of numbness starting in the middle finger of the left hand, extending to involve all the hand and the distal one half or two thirds of the forearm, without any recognized impairment of motor power. The numbness passed away about fifteen minutes later.

The progressive functional recovery of power in this patient may be illustrated by the recorded readings of the dynamometer used in testing the hand grasp. On September 28, the reading for the right hand was 65, that for the left hand 85; on October 5, the right hand 70, the left 85; on November 20, the right hand 95, the left from 85 to 90.

At the time of presentation there was still a residual weakness of the lower part of the right side of the face, the speech was still a little thick but much less so than formerly, the tendon reflexes were a little more active on the right than on the left, and there was a positive Hoffmann's sign on the right. The voluntary power of the extremities seemed to be entirely normal, however, and skilled movements of the hands were well performed on each side. There had never been any demonstrable sensory impairment at any of the several examinations. The patient was receiving antisyphilitic treatment, having started the course on Oct. 5, 1931.

Comment.—The mechanism of the production of such an attack may be explained on the basis of an interruption of the blood supply to the left internal capsule, but the relatively rapid recovery indicates that the stoppage of the blood supply was not of sufficiently long duration to produce serious degenerative changes in the fiber tracts. It is assumed that in this patient there exists a syphilitic disease of the walls of the blood vessels supplying the brain, with a resultant narrowing of the lumina of the vessels, and that the symptom complex is due to a temporary interference with the blood supply of the tissues in the distribution of the lenticulostriate artery, followed by at least a partial reestablish-

ment of the circulation within a short time. Such transient anoxemic states have been attributed by various authors to angiospasm of the cerebral vessels, and by others to a decrease in the volume of blood supplied to the tissues through sclerotic vessels during periods of lowered general blood pressure, as during sleep.

Dr. N. W. Winkelman (personal communication, repeated by permission) has proposed an explanation for transitory anoxemic symptoms in arteriosclerotic patients which seems applicable to this case. He believes that occlusion of a small blood vessel may give symptoms that indicate involvement of a much larger area because of the resultant edema and circulatory disturbance around the softened focus. With subsidence of the edema and partial circulatory adjustment the final area of involvement may be so small that the clinical symptoms may clear up almost entirely, especially in view of the tremendous compensation of the brain. Winkelman and Wilson (Segmental Cerebral Monoplegia of Vascular Origin, *J. A. M. A.* **86**:619 [Feb. 27] 1926) reported a case in which there was a severe degeneration of the lower motor area from vascular occlusion, which had produced a severe dysarthria that was temporary. Fifteen years after the cerebral lesion the patient came to the hospital with only the slightest hesitancy in speech, and yet at autopsy a goodly portion of the lower motor area was destroyed.

Solomon and Boshes (*Illinois M. J.* **60**:73 [July] 1931) reported the case of a man, aged 67, in whom repeated attacks of right hemiplegia associated with speech disturbances occurred, the attacks ceasing after three days of rest and the administration of coronary dilators. The blood pressure and the pulse rate dropped appreciably with the onset of an attack that they observed, and returned to the former levels as the symptoms cleared up a few minutes later. The changes in the blood pressure and pulse rate in their case may have influenced the rate of flow through the cerebral vessels. In my case the probable presence of a proliferative endarteritis and a perivascular infiltration and the fact that the patient still showed the residual signs of the hemiplegia are more in favor of Dr. Winkelman's explanation of the mechanism of transient hemiplegias.

DISCUSSION

Dr. William G. Spiller: The case is especially interesting because of the brief duration of the severe hemiplegia. It was associated with inability to speak and lasted about one hour, although weakness persisted in minor degree for a considerably longer time. A hasty observer might suspect an attack of hysteria in a colored woman, as hemiplegia has been recognized as produced by hysteria since the time of Charcot. The right hemiplegia associated with inability to speak, which seems to have been an aphasia, with much impairment of abdominal reflexes and facial paralysis of the upper neuron type indicates that the hemiplegia was organic.

Various explanations have been given for transitory hemiplegia. An attack lasting a certain number of hours or a few days is not uncommon in the aged and is attributed correctly to thrombosis of small vessels with some compensation by the collateral circulation, but an attack lasting a few minutes or an hour needs another explanation. Spasm of the vessels of the brain has been considered a cause of such attacks, and Spielmeyer has accepted the possibility of spasm of cerebral vessels. It is difficult to believe that highly sclerotic vessels may become spastic. Another interesting explanation has been given by Solomon and Boshes, who refer to the views of Fleming and Naffziger, that splanchnic dilatation may cause venous pressure to drop and this may produce decreased intracranial pressure so that blood passes rapidly from the arteries to the veins.

Dr. Melbourne J. Cooper: In connection with the changes in volume of the flow of blood through the cerebral vessels, Dr. Spiller has called attention to this factor in the case reported by Solomon and Boshes. Inquiry was made to elicit factors that might have influenced the blood pressure in my case at the time of the stroke. The patient was doing her usual housework, which was in no way strenuous; she had been up and about for several hours; she was not

overheated or excited: she had not overeaten, nor had she been drinking alcoholic beverages on that or the preceding day; she had been almost a total abstainer for several years. No abnormality of the circulatory system was found.

HYPERKINETIC MANIFESTATIONS IN A CASE OF CARBON MONOXIDE POISONING. DR. ALFRED GORDON.

This patient, 24 years of age, a carpenter, was found on March 10, 1931, under his automobile in a closed garage, with the engine not running but warm. His head was at the end of the exhaust pipe. He was unconscious, with both eyes open and the arms tightly flexed. At the end of thirty hours the physician in charge made the following observations: "The patient was semi-conscious, the pupils were unequal, reacting sluggishly to light. Cyanosis was evident all over the body. There was no rigidity of the neck. The patient was in a state of excitement, easily disturbed and difficult to control. The skin was dry. The heart was normal in size, free from murmurs, but the respirations were shallow. Blood examination showed hemoglobin 85, red cells 4,460,000; leukocytes 19,800; polynuclears 83 per cent. On the following day, a carbon monoxide test was done and it showed a heavy trace of the gas. The Wassermann test of the blood and spinal fluid was negative. The urine showed a low specific gravity and a heavy trace of albumin."

On March 16, I observed the following: extreme restlessness, rolling from side to side, unusual hyperesthesia all over the body and marked resistiveness. The least approach would throw the patient into a state of pronounced rigidity; the arms and legs would become hyperextended, just as one observes in experimental decerebration. The facial muscles were also rigid; the face would become set and the eyeglobes immobilized. Attacks of hyperextension occurred even when he was not touched. During the hour of examination, several such attacks were observed. During the patient's restlessness and during the process of rolling from side to side the limbs were also in extension. The extreme extension was observed when his body was manipulated. The tendon reflexes were somewhat increased, but there were no pathologic cutaneous reflexes. The pupils were unequal, the left slightly larger than the right. There was no nystagmus. The sphincters were not involved.

As to the neurologic symptoms, one was struck especially by the hypertonicity. The frequent attacks of extreme rigidity reminded one of laboratory experimentation on the brains of animals. Decerebrate rigidity is observed when the motor impulses are cut off from the prosencephalon. It has been shown that there is a tract arising in the cerebral cortex that has an inhibitory effect on rigidity. These fibers run in the mesial portion of the cerebral peduncles, cross and end in cells, fibers from which go through the middle cerebellar peduncles to the cerebellum and end in the superior vermis which, according to Sherrington, is the inhibitory center. Rigidity takes place when the inhibitory tract is severed and more especially so the thalamus.

Experiments on animals with toxic substances produce analogous phenomena. Buchanan injected into vertebrates from 5 to 10 minims (0.3 to 0.6 cc.) of 1 per cent mescaline and obtained hyperexcitability, paralysis and toxic rigidity (*Brit. J. M. Psychol.* **9**:67, 1924). Catatonic phenomena were observed following injections of 0.1 cc. of 1:1,000 suprarenal solution into a mouse and a cat. The same was observed with choline (DeJong), with phenol extract or with bulbocapnine. Catatonic motor symptoms, which sometimes follow carbon monoxide poisoning, have been associated with involvement of the striate bodies (Kant: *Arch. f. Psychiat.* **78**:365, 1926). This is particularly seen in experimental bulbocapnine poisoning. The majority of experiments, however, prove that extensor phenomena are observed not so much when the motor control of the prosencephalon is interrupted as when the important centers of the midbrain are put out of function.

In the case described there was also a phenomenon that is observed in extrapyramidal lesions, namely, hypertonicity of the limbs on active or passive movement. The pathologic substratum in extrapyramidal cases is predominantly a

lesion in the lenticular nucleus and especially the globus pallidus. The rôle of the latter in the pathogenesis of tonus is well known; it constitutes one of the most important centers for the regulation of tonic functions. Various opinions have been expressed concerning the predominance of pallidal lesions in diseases of the midbrain. Kolisko, for example, incriminated the arterial supply of the pallidum; he believed that the absence of anastomotic branches in this portion of the lenticular nucleus, as well as the curved and bent form of these very small blood vessels, predisposes them to congestion, stasis and finally thrombosis, thus creating a necrotic state of the surrounding tissue (*Beitr. z. gerichtl. Med.*, 1914, vol. 2). Other investigators (C. and O. Vogt, Stewart and Grinker) believe in a direct influence of carbon monoxide or in a special physicochemical affinity of the latter for the pallidal tissue.

Considering the anatomic peculiarities of the pallidal arterioles and bearing in mind the anoxemia that is caused by the fixation of carbon monoxide on the hemoglobin, one is justified in accepting the view that the damage of the pallidal tissue is due to the unfavorable nutrition of the nerve tissue. If one adds a third pathogenic element, namely, a vasomotor paralysis of these arterioles for the existence of which there is some physiologic proof (cyanosis, herpetic eruptions, localized edema and discoloration of the skin), the effect of carbon monoxide on the cerebral elements becomes self-evident.

This case was recorded chiefly because of the somewhat unusual neurologic manifestations, the rapidity of its course and the great damage caused by carbon monoxide.

MERALGIA PARAESTHETICA. DR. ROSS H. THOMPSON.

The interesting feature of this case is that it presents an isolated involvement of a single peripheral nerve, the external cutaneous nerve of the thigh. Bernhardt first described the condition in 1895, but Roth, a Russian, later in the same year established it as a clinical entity and gave it its name, meralgia paraesthetica. According to Roth, there are three points in the course of the nerve, from its origin in the second and third lumbar nerves to its distribution to the skin of the lateral aspect of the thigh, for purely cutaneous sensation, at any one of which it may be subject to trauma, such as pressure: (1) as it emerges from beneath the psoas muscle, (2) as it passes over the ilium to emerge from the pelvis, near the anterior superior spine, and (3) as it passes into the fascia lata of the lateral aspect of the thigh. Among the factors of mechanical pressure may be mentioned trusses, belts, tight bands and corsets.

The patient, a man, in the service of Dr. Spiller, had an oval area of numbness on the outer side of the left thigh just above the knee. It appeared first sixteen months prior to presentation. The area felt as though it had been frozen. At the time he was first seen in July, 1931, he was fairly stout and wore a thin belt tight and low over the hips, exerting pressure in the region where the nerve passes over the crest of the ilium. There were no disturbances other than sensory and no atrophy, and the tendon reflexes were normal.

Within this oval patch there were also objective disturbances of sensation: hypesthesia to light and deep touch, hypalgesia to pinprick and thermhypesthesia to heat and cold. Sensation for deep pain was not disturbed, nor was vibratory sensation.

The patient was advised to discontinue wearing the belt and to have the thigh massaged two or three times weekly. He has not carried out this treatment consistently, so that over a period of five months there had been no improvement. The part felt just the same.

According to Stookey, the etiologic factors in the production of true meralgia paraesthetica are the abnormal position and angulation of the external cutaneous nerve as it leaves the pelvis. He was of the opinion, in 1928, that the proper treatment is section of the nerve to produce a permanent anesthesia, which is

preferable to the discomfort of the disease. Dr. Spiller reported a case in 1898 and suggested section of the nerve in cases that did not respond to the usual therapeutic measures.

Another patient had a similar condition in both thighs due to an occupational pressure. He was a locomotive engineer, fairly stout, who found it necessary to distort his body considerably and press each side in leaning out of the windows of the engine cabin. All of his symptoms, which were the same as in the first patient, promptly disappeared when he discontinued the stress and pressure on leaning out of the window.

DISCUSSION

DR. W. B. CADWALADER: In a patient in Dr. Spiller's service with this syndrome the symptoms were attributed to a large mass of varicose veins in the inguinal region, which compressed the nerve trunks.

DR. H. I. GOLDSTEIN: In the *American Journal of the Medical Sciences* (**162**:720 [Nov.] 1921) I published a paper on meralgia paraesthesia (Roth's or Bernhardt's disease), and reported six cases. One patient, a man, aged 45, was a dealer in bottled goods; he had delivered box and case goods to customers from a wagon for a number of years. I believe that the constant pressure and repeated trauma caused by the heavy boxes against his side brought on his trouble. The condition became aggravated on walking or standing. Studies of the blood and urine gave negative results. This was an occupational case. Another case was in a woman, aged 44, who worked in a restaurant. She wore a long, tight corset, the constant pressure of which may have caused the trouble. She also had a 4 plus Wassermann reaction of the blood and was passing through the menopause. Four probable factors — occupation, pressure from a corset, syphilis and the climacterium — may have been operating in this case.

DR. ROSS H. THOMPSON: I purposely did not mention the patient's occupation because I thought it had no etiologic reference to the condition of the lateral cutaneous nerve. He is employed in the physical therapy department of the University of Pennsylvania Hospital. In looking for a cause, the constriction of a tight, thin belt worn low over the hips, exerting pressure at the point of emergence of the nerve from the pelvis, was assumed to offer a satisfactory explanation. This disease has been attributed to many factors, including disease of the spinal cord, vertebral and pelvic disease, alcohol, rheumatism, pressure and other trauma. Most neurologists, however, hold to the view that disturbances of the peripheral nerve alone should be taken into consideration.

TETANUS OF THE HEAD WITHOUT A WOUND OF THE HEAD. DR. A. M. ORNSTEEN, DR. N. S. YAWGER and DR. J. A. WAGNETC.

A boy, aged 14, entered the hospital with a complaint of stiffness of the neck, jaw and back. On Sept. 23, 1931, he had stiffness and pain in the jaw and glands of the left side of the neck. The glands were swollen on the left side. He went to school as usual and had no treatment, but on September 25 there was marked stiffness of the neck and head. The head was twisted to the right. A physician prescribed for him and sent him to bed. At this time the head was twisted to the right. There was no fever or acceleration of the pulse. Two days later, the patient could not swallow, but nothing was found to account for the difficulty in swallowing. The condition became progressively worse; the stiffness extended down to the back, and there was more retraction of the head. There was no history of a wound or abrasion of the cutaneous surfaces. He had had a sore throat at times, measles and whooping cough, and an appendectomy had been performed six years before examination.

Examination showed a well nourished boy lying quietly in bed, with the head retracted and the knees flexed, in no apparent discomfort. The left eye was somewhat swollen. The left pupil was smaller than the right and reacted less promptly to light. Both pupils reacted in accommodation. There was distinct photophobia; the left palpebral fissure was narrowed, and the forehead moved

freely. There was inability to open and close the mouth fully. The left angle of the mouth could not be elevated as much as the right. The tongue was protruded with great difficulty in the midline. Examination of the throat was impossible. The cervical glands were not enlarged. The chest and heart were normal. The liver and spleen were not enlarged. The abdomen was tense but not distended. The cremasteric reflexes were present. There was marked rigidity of the neck and back. The lower extremities suggested spasticity; the upper extremities were freely movable. The reflexes in the upper extremities were prompt and equal. The knee jerks were prompt, increased and equal. There was no Babinski, Oppenheim or Gordon sign, but a suggestive ankle clonus was present. Kernig's sign was moderate on the right side. Lumbar puncture on September 30 gave clear fluid under pressure; there were no cells, and a culture gave negative results.

While at home, on trying to speak the patient became cyanotic and dyspneic and frothed at the mouth. A similar attack was noticed at the hospital only on two occasions, each time when trying to speak or in opening the mouth. The attack was relieved by epinephrine. The patient was treated with antitetanus serum, being given 10,000 units intraspinally, intramuscularly and intravenously. After this, the intravenous method was used chiefly, in doses of from 20,000 to 30,000 units daily; all told, about 160,000 units were given in from six to seven days. On the eighth day, the patient had the usual reaction, with glandular enlargements throughout the body. About the third day, the rigidity started to lessen and the patient felt better. At no time were there any vesical or rectal symptoms.

Five lumbar punctures were made; the pressure varied from 40 to 18 mm. of mercury at the last puncture. Three samples of spinal fluid were cultured; all gave negative results and an alkaline reaction; sugar and a normal amount of globulin were present. One lumbar puncture showed no cells; others, 10 per field and 30 per field, and the last no cells. Two examinations of the eyegrounds were made, one showing evidence of choked disks and the other showing the condition improved but still present. A blood count on admission showed: hemoglobin, 80 per cent; 5,050,000 erythrocytes; 14,400 leukocytes, of which 82 per cent were polymorphonuclears. The white cells dropped later to 9,000.

The temperature was irregular, varying from 100 to 103 F. up to the sixth day of the disease, gradually reaching normal on the seventeenth day of the illness. The pulse rate ranged from 130 to 140, and was rather proportionate to the rise in temperature. The respirations ranged from 30 to 50 per minute.

There was no history of injury at any time, except that the patient had had a recent injury on the right elbow which was still covered with a scab. He informed us that he had contracted this about two or three weeks before admission while roller skating, striking the elbow on the curb.

DISCUSSION

DR. A. M. ORNSTEEN: The interest in this case is based on the fact that the so-called tetanus of the head of Rose developed without a facial wound, and because of the facial hemiplegia and dysphagic symptoms. The development of the symptom complex was: Stiffness and pain in the masseter regions, with stiffness of the neck, followed in forty-eight hours by fever and inability to open the mouth. In rapid progression followed dysphagia, weakness of the left side of the face, photophobia, retraction of the head, stiffness of the back and general tonic spasms brought on by attempts to swallow, much like a hydrophobic case. Respiratory embarrassment was experienced during the acute stage to the extent of several attacks of dyspnea with cyanosis. There was a left facial hemiparesis of the kind described as characteristic of tetanus, namely, the appearance of paralysis with a contracted state of the affected muscles at the same time. This gave the appearance of a narrowed palpebral fissure and a contracted corner of the mouth on the affected side, much like the appearance of an old incompletely recovered facial palsy with secondary contractures. There were marked "lock

jaw" and severe nuchal rigidity, with retraction of the head, stiffness of the back and a moderate Kernig sign on each side. Weakness of the left external rectus muscle and pupillary inequality, with good reactions, were observed. There was a moderate degree of papilledema, which was not measurable. Because of the trismus it was not possible to examine carefully the palatal and pharyngeal muscles. Examinations of the spinal fluid gave essentially negative results, thus ruling out meningitis. The limbs were not deficient in motor power, the tendon reflexes were prompt and moderately hyperactive, and the plantar reflexes were of the flexor type. There was no evidence of local tetanus in the neighborhood of the wound. Treatment was instituted on the second day after admission to the hospital, which was eight days after the onset of symptoms. The patient received within one week a total of 154,000 units of tetanus antitoxin intravenously and intramuscularly. The temperature ranged from 99 to 103.8 F., and was of remittent character, whereas the pulse rate was erratic and was generally faster than the ordinary relation to the temperature. Other laboratory studies were of no diagnostic value.

The boy made a perfect recovery within four weeks and is today without symptoms. Therefore, it can be said that tetanus may result from a superficial wound and that tetanus of the head does not necessarily result only from a wound in the neighborhood of the facial nerve. The reported case can be spoken of as one of hydrophoboprosoplegic tetanus.

TRAUMATIC SENSORY APHASIA WITH RECOVERY. DR. WILLIAMS B. CADWALADER.

The object in presenting this case is to show that profound word deafness and word blindness may occur from an injury of the skull, probably with meningeal hemorrhage on the left side of the brain in a right-handed person, and that complete recovery may take place without operation.

A woman, aged 24, a stenographer, was admitted to the hospital on Feb. 2, 1931, while unconscious, immediately after an automobile accident. She recovered consciousness on the second day, and it was then noted that there was slight weakness of the right side of the face and the right upper and lower limbs. She was unable to express herself in words and did not seem to understand what was said to her.

Roentgen examination revealed a suture fracture between the frontal and parietal bones, extending posteriorly into the right parietal region, high in the cranial vault. Ocular examination showed that the disks and movements were normal.

On the fourth day after the injury, there was a slight clonic convulsion involving only the right side of the face and the right upper limb. On February 18, the patient was fully conscious, sitting up in bed and normal in appearance, but the right side of the face and the right upper and lower limbs were still slightly weaker than the left. The tendon reflexes were increased on both sides, but were slightly greater on the right. In attempting to speak the patient uttered sounds that were entirely unintelligible. She could not formulate a single word and when questioned would nod her head in a meaningless manner. She did not understand even the simplest phrases and did not carry out the simplest commands. When given a pad and pencil and asked to write she seemed to understand what they were intended for, but in attempting to write she made scrawls on the paper in an awkward manner and did not formulate a single letter or figure. She would then stop and look up in an inquiring manner and utter sounds that were unintelligible as words. Written words or figures were meaningless to her. She did not add or count, but copied a few simple letters, though imperfectly. Her inability to perform these acts was not due to incoordination or to muscular weakness. The symptoms gradually became less marked, and she recovered the ability to speak, write and read.

When examined on April 21, she pronounced words fairly well but occasionally made mistakes. She could write spontaneously and also from dictation, but did so slowly, and this seemed to require considerable mental effort. On one occasion she said that she required one hour to compose and write a short letter.

On May 20, all these disturbances seemed to have entirely disappeared and her ability to carry on a conversation, to read and to write appeared to be normal; on July 1, she returned to her former occupation as a stenographer, where she has been occupied since. Her employer has reported that her work in this capacity has been entirely satisfactory.

It seems most probable that as a result of the injury the left cerebral hemisphere was damaged, including the first temporal convolution and the region of the angular gyrus, since the symptoms were those of complete word deafness and complete word blindness, and perhaps partial apraxia.

In 1924, Gerstmann (*Wien. klin. Wchnschr.* **37**:1010, 1924) described a syndrome that he called "finger agnosia"; he defined this as consisting of an isolated disturbance in recognition, naming and selection of the individual fingers of the patient, of the hands of others or of hands drawn on a blackboard. Associated with the finger agnosia were agraphia and acalculia. In his first case the patient had had a stroke one year previous to examination. The finger agnosia did not develop until much later. In 1927 (*Ztschr. f. Neurol. u. Psychiat.* **108**:152, 1927) and 1930 (*Deutsche Ztschr. f. Nervenh.* **116**:46, 1930), the same author reported two additional cases.

In 1926, Potzl and Hermann (Ueber die Agraphie und ihre lokal diagnostischen Beziehungen, Berlin, S. Karger, 1926) reported one case, and in the same year, Hermann and Kerschner (*Med. Klin.* **22**:1821, 1926) described another case; in 1930, Lange (*Monatschr. f. Psychiat. u. Neurol.* **76**:129, 1930) reported a case, making a total of six cases.

In Gerstmann's cases there were no aphasic, apraxic or other agnostic symptoms, but merely an elective agnosia of the fingers, with isolated agraphia.

In the case described by Hermann and Potzl there was a tumor in the parieto-occipital convexity, and pure agraphia dominated the clinical picture; but as the injury of the tumor spread other symptoms and finger agnosia developed. In this case the tumor was found in the right side of the brain. In Gerstmann's cases and all other cases reported the lesion was on the left side, and in these cases there was softening and not a tumor. In Hermann and Kerschner's case the finger agnosia was used as a focal diagnostic sign; operation confirmed this, showing a cyst at the transition between the gyrus angularis and the second occipital convolution. This patient recovered.

In the case I have presented an attempt was made to determine whether finger agnosia and acalculia were present, but because of the patient's inability to converse and to understand commands the results were so confusing that I do not think they were sufficiently clear to draw conclusions. At present this patient presents no symptoms whatsoever.

DISCUSSION

DR. BERNARD J. ALPERS: Were any observations made on the mode of return of speech and what functions returned first?

DR. W. B. CADWALADER: In answering Dr. Alpers' question as to the order in which the return of speech came about, I must say that I could not follow it. I saw the patient only at irregular intervals of a month or so.

THE LOCALIZATION OF INTRACRANIAL TUMORS BY THE BÁRÁNY METHOD. DR. R. J. HUNTER.

Although the localization of intracranial tumors has been greatly advanced by the newer methods of ventriculography and encephalography, many cases still remain obscure. Therefore, one must frequently employ every diagnostic measure that will help to discover the area that is involved. The reactions resulting from stimulation of the vestibular nerve and its intracranial and peripheral pathways are important. The exact tract traversed by all the fibers is not known. The Bárány tests must be considered in connection with other physical signs and like all diagnostic aids require careful observation and a certain skill in eliciting them,

making the personal equation of the observer an important factor. I have divided the cases roughly into supratentorial and infratentorial lesions. The former only will be discussed here.

In patients with supratentorial lesions the Bárány reactions may be normal, but they are more likely to be hypersensitive. Several points concerning the eyes are important. Spontaneous nystagmus is often absent, but when present it can frequently be changed in direction or increased in intensity by proper stimulation. During the caloric test one must look for these things: lowered threshold and a long duration of nystagmus, or a lowered threshold and conjugate deviation of the eyes may be present at the same time. By lowered threshold is meant that the nystagmus is induced more quickly than usual. The normal time for reaction through the vertical canals is from forty to fifty seconds with water at 68 F. When the inhibition is removed the nystagmus, once induced, lasts a long time. Normally it stops in about one minute. Two and a half minutes are considered an abnormal duration. Vestibular nystagmus is an alternate movement of the eyes to and fro. There is a slow movement in one direction, followed by a quick movement in the opposite direction. The two eyes usually work in unison, but dissociated nystagmus does occur. Conjugate deviation of the eyes, in which both turn to the side of the slow component or in which there are desultory quick jerks with a slow long pull in the direction of the slow phase, is often said to be an indication of a supratentorial lesion because the quick component of nystagmus is supposed to be controlled by the cerebrum. When cerebration is depressed, the slow or cerebellar pull is not compensated for and conjugate deviation results. A study of the ocular pathways shows that conjugate deviation may also be caused by pontile and many other lesions. It has been produced by stimulation of parts of the frontal, temporal, occipital and parietal lobes and by destructive lesions in the internal capsule, bulb, pons and cerebellum. When present spontaneously, it may be intensified by a cold caloric stimulation of the side toward which the eye is turned. In destructive lesions it is toward the side of the lesion in cerebral cases and away from the lesion in cerebellar cases. The reverse is true of irritative lesions. De No is quoted as saying, "in man a lesion of the vestibular nuclei causes a conjugate deviation of the eyes only if the lesion is seated in Fuse's nucleus (near the sixth nucleus) and the deviation is toward the side opposite the lesion." He also said, "While the cerebral cortex and thalami are not necessary for the production of the quick phase of nystagmus, there is a point in the brain stem where the regulating system for ocular movements is joined by the oculomotor nuclei; if this point is destroyed the rapid component of nystagmus is lost." Another thought about the quick phase is that the lower reflex center may be actuated by an overflow from the center for the slow pull. Conjugate deviation seen after vestibular stimulation may be caused by reflex action, revealing a latent lesion of one of the numerous centers for the associated movements of the eyes. It may be either an irritative or a destructive lesion, in the vicinity of one of the aforementioned centers. A lesion may not cause oculogyric spasm or paralysis, but it may interfere with an oculogyric center to such an extent that vestibular stimulation will bring out the latent defect. Voluntary lateral movements of the eyes are governed by the second frontal convolution and may be paralyzed while reflex movement from vestibular stimulation can still be produced. If it is true that the quick component is due to the conscious effort to fix the object, one can understand that an unconscious patient will show merely a pull in one direction resulting in conjugate deviation. This is seen when the labyrinth is stimulated during anesthesia. Dande has located consciousness "at some point in the left cerebral hemisphere and within the blood supply of that part of the anterior cerebral artery lying above the anterior third of the corpus callosum." Alpers and Grant reported Bárány signs of a supratentorial lesion in a tumor of the corpus callosum. They stated that inability to concentrate and keep the attention focused is one of the outstanding symptoms of a tumor of this area. Unfortunately, they did not state what the Bárány signs were, but one could expect that inattention would lead to conjugate deviation. Brunner and Hoff

observed a case with a cyst over the angular convolution which showed conjugate deviation to the side of the lesion. They quoted Pötzl as authority that a center exists here which receives vestibular stimuli that are sifted, suppressed or compensated for by ocular movements. This center may give conjugate deviation if its activity is suppressed, and it occurs to me that this center, when very active, may be the cause of a lessened response to rotation and caloric tests, which have been found experimentally in rats subjected to frequent rotations and which I have noted in a pirouette dancer and described in an article on the falling reaction in acrobatic aviators.

Past pointing may be present spontaneously in supratentorial cases. The direction can frequently be changed or the amount of error increased by appropriate stimulation. This is not the case in most cerebellar lesions in which the error persists regardless of how much one stimulates the ear. The location of the lesion according to the direction of past pointing in supratentorial cases is not well understood. It has been my practice to try spontaneous past pointing in both the vertical and the horizontal plane, because I have found that some patients touch spontaneously in one plane, but not in the other. One should note also at the same time whether the arm is raised or lowered in an abnormal way. Failure to past point after stimulation may also be present. If so, one can often make the area past point by irrigating another canal or the opposite ear. Hearing is usually normal, except in some lesions of the temporal lobe in which there may be hallucinations of hearing. Vertigo and nausea are likely to be more marked than normal in supratentorial cases. Fisher reported that susceptibility to stimulation was absent in patients with lesions of the temporal and occipital lobes.

Seven case histories of tumors of the brain were taken in numerical order from the files at the Philadelphia General Hospital in which a Bárány opinion had been given by me and a neuropathologic study of the brain and stem had been made by Dr. Winkelman and Dr. Riggs of the division of neuropathology. The vestibular tests will be discussed to see why a given opinion was reached. I shall point out that in several of the cases, although the diagnosis, which was of course merely from the Bárány standpoint, was correct, it was made with a knowledge of the general condition of the patient, and could have been given another interpretation. I shall also point out that in some cases an incorrect localization would result from the Bárány reaction alone and shall discuss my mistakes. Of these, three lesions were diagnosed as supratentorial.

Case 1.—The patient was seen in Dr. McConnell's service and gave a history of loss of memory for six months and attacks of vertigo for one month, with unconsciousness and great frontal headache. He was disoriented; his memory was poor and his gait staggering. Neurologic examination at first glance suggested a lesion of the frontal lobe to Dr. McConnell when he saw the patient in consultation in the psychiatric ward, but on transfer to his service and after more careful examination he believed that the lesion was probably cerebellar. He ordered a ventriculogram, which greatly helped to clear the diagnosis. It showed great distention of both ventricles and a possible blocking at the aqueduct of the fourth ventricle. This information confirmed Dr. McConnell's suspicions, and he made a diagnosis of a right cerebellar tumor. Autopsy showed a diffuse tuberculoma in the right cerebellar hemisphere, extending into the right cerebellopontile angle, with the corpus callosum as thin as paper and diffuse internal hydrocephalus.

On June 16, 1930, a Bárány test was made. I was unable to complete it on account of the physical condition of the patient. He obeyed commands with either ear occluded, but I was unable to determine accurately the amount of hearing. There were spontaneous rotary nystagmus to the right and some horizontal nystagmus to the same side. In a cold caloric test the right ear showed prompt and active ocular responses, with a tendency to conjugate deviation of the eyes to the right. In the left ear I could not be sure of any change in the spontaneous rotary nystagmus, but the amplitude and rapidity of the spontaneous horizontal nystagmus were promptly and markedly increased and continued for two and one-

half minutes before resuming the rate and amplitude that were present spontaneously. Owing to the lack of cooperation, I could not try past pointing through either ear, but in each case the patient showed deviation of the arms in the proper direction and I concluded that he received a stimulation through all canals. I gave the opinion that the Bárány tests pointed to a supratentorial lesion, because of the hypersensitive reactions in both ears. I ventured to suggest that it was on the left side, because there was rotary nystagmus to the right, which I thought would indicate disease of the left cerebral vestibular tract.

Autopsy Record.—The right cerebellar hemisphere was larger and softer than the left. This was the site of a diffuse nodular growth which blended into normal tissue. This mass covered the lower surface of the hemisphere and surrounded the periphery in an irregular band. The superior surface was about half covered with the mass, which spread across the midline and formed a small nodule on the left hemisphere. The growth was yellow and soft and showed some areas with central necrosis. On the right side the growth extended into the cerebellopontile angle. A section of the brain showed enormous internal hydrocephalus, limited to the lateral ventricles. The corpus callosum was as thin as paper. A microscopic examination of the tumor showed it to be a tuberculoma.

I was justified in this case from a Bárány standpoint in making a diagnosis of a supratentorial lesion, if one can depend on the accuracy of the findings that all canals were functioning, but on retrospect, I find: 1. The patient was too sick to be examined satisfactorily. 2. No impulse may have gone through the left vertical canals. The presence of spontaneous nystagmus to the right made it hard to determine this point after irrigating the left ear. 3. The evidence of normal past pointing was not complete, as the patient would not try to touch the examiner's finger. I measured the impulses by the drift. This is good evidence, but, of course, is not as satisfactory as actual past pointing. However, it is not necessary to go this far afield to support the diagnosis. The fact that the enormous internal hydrocephalus, limited to the lateral ventricles, involved the corpus callosum and that the tuberculoma of the right cerebellar hemisphere was rapidly growing might account for the fact that eight days before death, when the Bárány test was made, there were hearing, nystagmus and muscle drift. It is strange that, although the lesion was in the right cerebellum, the right ear responded better than the left. The same physical condition of the brain that gave the Bárány symptoms of a supratentorial lesion gave other symptoms of the same character, leading the neurologists to favor the diagnosis of a frontal tumor until a ventriculogram showed that the lesion was probably posterior. There was also the conjugate deviation of the eyes to the right, after irrigation of the right ear, which would point to a left cerebellar or a right cerebral dectructive lesion. The involvement of the corpus callosum may have caused the deviation of the eyes. Another important feature of the Bárány test in this case was the length of time the nystagmus continued after stimulation was discontinued. Not only did nystagmus appear promptly, but it continued a long time after irrigation of the left ear (two and one-half minutes). I interpreted it as a lack of cerebral control. This case would support the theory of De Stella that the functioning of the labyrinth is independent of the cerebellum.

Résumé.—A right cerebellar tumor was incorrectly diagnosed by the Bárány method as a left supratentorial lesion after one examination. The neurologists made the correct diagnosis.

Case 2.—D. D., a white man, aged 39, who was admitted to the hospital on March 12, 1929, in the service of Dr. McConnell and Dr. Weisenberg, had irrelevant family and past medical histories. Three weeks before admission, he stooped over and hit the left frontal region. He said that the blow dazed him, but he did not lose consciousness. Following the blow he had severe frontal and occipital headache almost constantly. He had been in bed for two weeks before admission to the hospital. General physical examination gave negative results. Mentally the patient showed an attitude of defiance. His wife said that his personality had not changed following the blow, but as the case progressed it was

noted that he did not appreciate the seriousness of his case but laughed about it. A tendency to disorderly personal conduct developed. The patient was oriented as to time, place and persons. As the disease advanced he became drowsy and finally (on August 11) stuporous. The pupils were equal and responsive to light and in accommodation, but both eyegrounds showed definite blurring of the nasal portion. There were hemorrhage and edema of the disk. On March 15, 1929, Dr. Holloway reported the ocular movements full; the right pupil was larger than the left; both disks showed choking, which had progressed by April 29 to from 4 to 5 diopters. The temporal borders were hazy, but not completely obscured. The deep reflexes were equal and normal in the upper and lower extremities. There were no pathologic reflexes. Movements of the extremities were well performed, but synergic movements seemed to be better on the right side. There was some definite incoordination. The gait was rolling. The left side became weaker and finally was paralyzed, with sensory loss and left facial weakness on April 25. A diagnosis of neoplasm of the right frontal lobe was made. Dr. Fay operated on the right frontal temporal region on April 25 and found a glioma. The spinal fluid was clear and showed a pressure of from 14 to 18 mm. of mercury. The Wassermann reaction was negative. Death occurred on August 11.

The Bárány examination in detail, on March 22, 1929, was as follows. The patient showed spontaneous horizontal nystagmus to the right, which was decidedly lessened on looking to the left. There was no spontaneous past pointing in either the horizontal or the vertical plane. The cold caloric test of both ears showed normal hearing to the whispered voice. All pathways transmitted impulses normally and gave a proper reaction for nystagmus and past pointing, except that the reaction in the right ear began in twenty seconds (normal, forty-five seconds). The direction of the spontaneous nystagmus which the patient showed could be changed by stimulation of the ear. He showed a normal amount of nausea.

I again examined the patient on April 23, and found essentially the same symptoms as on the first examination. The patient developed rotary nystagmus in eighteen seconds after irrigation of the right ear with cold water. On irrigation of the left ear he developed rotary nystagmus in twenty-four seconds. The horizontal nystagmus and past pointing were normal. He was distinctly hypersensitive. The spontaneous nystagmus, which was the same as on the first examination, could be changed in direction by stimulation of the ear. Nausea was marked after irrigation of either ear. Hearing was normal.

Accordingly I reported: The examination suggests that the lesion is (1) supratentorial, or (2) that the factor causing the hypersensitive reaction is the cause of the nystagmus. The nystagmus is due to an irritative lesion and not to an imbalance by having one pathway cut off. The fact that all pathways are open to stimuli suggests that the lesion is not in the posterior fossa. I suspect that the lesion may be frontal. Two days later, subtemporal decompression was done by Dr. Fay, whose diagnosis was glioma of the right temporal lobe.

Pathologic Description.—An enormous fungating tumor occupied all but the frontal and occipital poles of the left hemisphere. The mass began 2.5 cm. beyond the frontal poles and extended back to the calcarine fissure. It covered the lateral and inferior surfaces of the hemisphere, destroying the entire cortical tissue, and on the orbital surface extended mesially to the interpeduncular space. The corpus callosum on the right side was thin, especially in the middle third, where it showed invasion by tumor tissue at what corresponded to about the level of the mesial nucleus of the thalamus. On section, the structures of the right side of the brain had been pushed to one side by the neoplasm. The ventricles were not dilated. The entire basal ganglia of the left side were destroyed. The tumor extended into and practically filled the left lateral ventricle. The wall of the anterior horn of the left ventricle was involved in the tumor. The microscopic description was spongioblastoma multiforme.

Reviewing the findings, it is to be noted that the correct localization in the case was based partly on the knowledge that the patient had a tumor of the brain. I should not have used the term irritative lesion. It would have been better to

say the higher centers controlling the vestibular sense are involved, because I diagnosed the case as one of a destructive lesion. The same Bárány reactions might be found in food poisoning, migraine or foci of infection.

CASE 3.—A. Y., a woman, aged 40, was admitted to the hospital on April 11, 1927, in the service of Dr. McConnell. She had been showing mental changes for two years before 1925. There was no history of encephalitis or acute illness. She was slothful, sat in one spot for hours without moving and slept a great deal. Loss of memory was noted, but there was no delirium or hallucinations. She had been shy and retiring, but had become boisterous, insolent, free of speech and jocose and careless in dress. Her speech was of a drawling, slurring, scanning type. There was intention tremor of the tongue, lips and upper extremities. The disks showed an early blurring in the left eye and choking in the right eye. The eyegrounds were well defined. Weakness of the left side of the face and of the left hand and arm was present. The arms were spastic, and there was clonus in the right arm and wrist. All deep reflexes and sensory changes were absent. Sphincter ani and vesical control had been gradually lost. The gait was staggering. Verbal and tactile perseveration were present. The serology was normal; the spinal fluid pressure was 234 cm. of water, and on May 24, 350 cm. of water. The cell count was 10; there was a trace of globulin; the colloidal gold curve was normal.

On May 26, I examined the vestibular apparatus. There was no spontaneous nystagmus. There was spontaneous past pointing to the right, with the right arm moving in a vertical plane. This was unchanged with the head either 30 degrees forward or 60 degrees backward. The patient past pointed under with the right arm moving in a horizontal plane; she touched in all positions with the left arm. During a caloric test of the right ear, rotary nystagmus was noted in forty-five seconds. She past pointed under through the vertical canals, with both arms moving in the horizontal plane. When the head was placed back, a well marked horizontal nystagmus developed. She past pointed to the right with both arms, the spontaneous past pointing error of the right arm having been increased by the irrigation. A caloric test of the left ear showed rotary nystagmus in fifty seconds. When the head was placed backward, horizontal nystagmus with wide excursions was noted, followed by conjugate deviation of the eyes to the left. Past pointing through all three canals was normal. The nystagmus having been of an unusual nature, I examined her a second time and stimulated the left horizontal canal only. In eighteen seconds the same reactions as those previously noted were seen.

My conclusion, given to Dr. Riggs personally, was that there was a supratentorial lesion, probably in one of the frontal lobes, but I was unable to say in which.

On May 31, 1930, a subtemporal decompression was performed by Dr. Grant. The pathologic report showed that an enormous neoplasm, springing from the falx in the midline, occupied the greater part of the anterior two thirds of the left frontal lobe and one half of the right frontal lobe. It measured 8.5 by 7.5 by 6.5 cm. and shelled out easily, leaving an enormous depression. The neoplasm itself was firm. The tumor had destroyed the anterior portion of the corpus callosum by pressure and had also destroyed the anterior horns of the lateral ventricles. There had been a spontaneous decompression through the floor of the sella. A microscopic report showed the tumor to have been an endothelioma. The interesting feature of these findings was that conjugate deviation to the left was seen only on stimulation of the left horizontal canal and not of other canals. This might have led me to make a diagnosis of a left-sided cerebral lesion. There should be noted: the destruction of the anterior portion of the corpus callosum; that the spontaneous past pointing to the right with the right arm was increased by irrigation; the abnormality on stimulation of the pathway of the right vertical canals to the left arm (viz., she past pointed under instead of above).

Summary.—1. Vestibular examinations are important in supratentorial lesions. Even a normal reaction is of diagnostic value in ruling out an infratentorial

lesion. 2. Spontaneous nystagmus and past pointing can more often be changed in direction by irrigation of the ear than in infratentorial cases, and are less frequently seen. 3. Nystagmus, past pointing, vertigo or nausea more marked than normal is often seen after stimulation. 4. The normal inhibition of nystagmus after stimulation of the ears is frequently absent or lessened, resulting in prolonged duration of nystagmus. 5. The reaction time of nystagmus is shortened in many cases. Nystagmus appears more quickly than normal in response to a standard stimulus. 6. Conjugate deviation of the eyes may indicate either a supratentorial or an infratentorial lesion.

DISCUSSION

DR. FRANCIS C. GRANT: In the last several years, in Dr. Frazier's service at the University Hospital, our success with and our reliance on the Bárány test has decreased. The reason for this is I think, in part at least, the personal equation in the carrying out of these tests, which is, as Dr. Hunter stressed, an extremely important factor. Five or six years ago, Dr. Fisher was extremely interested in this work and carried out the tests in practically all of our cases of tumor of the brain. At that time we reviewed and published (Grant and Fisher: One Hundred and Sixteen Verified Intracranial Lesions with Report of Vestibular Test Findings, *J. f. Psychiat. u. Neurol.* **34**:3 and 113, 1926) our results with the Bárány tests in 116 verified and seventy-three unverified tumors of the brain. In the verified group were thirty-three cerebellar tumors, twenty in the cerebellar hemispheres and ten in the vermis, and in three instances no cerebellar tumor was found at operation. In the cerebellar hemispheric group of twenty tumors, the Bárány reactions correctly localized the lesion in seventeen patients; in eight of the ten lesions in the vermis and in the three cases in which no tumor or evidence thereof was discovered at operation the tests stated that no tumor was present. The reactions gave misleading information in three of twenty hemispheric cerebellar lesions and in two of ten midline cerebellar lesions. In thirty-five cases of verified tumors of the cerebellopontile angle, the Bárány tests agreed with the operative findings on thirty occasions. In six of these thirty-five cases the reactions afforded the most positive early evidence of the presence of the tumor. Of thirty-five cases of cerebral hemispheric lesions, in thirty-two the Bárány reactions indicated that a lesion of the brain was present, and in nineteen lateralized it correctly. In three instances the tests were proved incorrect. In thirteen cases of midline cerebral lesions the reactions were interpreted as indicating the position of the tumor in six instances, while in the remaining seven, as a result of the tests, the posterior fossa was excluded as a possible site for the tumor.

As a result of this study, the following conclusions were reached: 1. Cerebellar lesions give the following group reactions: (*a*) poor movements of the pelvic girdle; (*b*) marked tolerance to vestibular tests; (*c*) interference with vertigo and pastpointing responses; (*d*) predominant signs of intracranial pressure. 2. Lesions of the cerebellopontile angle give the following reactions: (*a*) totally impaired eighth nerve on the side of the lesion in both the cochlear and the vestibular divisions; (*b*) loss of function of the vertical canals on the opposite side, but good function from the horizontal canal, with good hearing. 3. Supratentorial lesions, midline or hemispheric, give the following reactions: (*a*) marked susceptibility to vestibular stimulation; (*b*) conjugate deviation of the eyes to the side of the lesion; (*c*) exaggeration of the nystagmic responses over vertigo and past-pointing responses; (*d*) no evidence of a cerebellar hemispheric lesion, though increased intracranial pressure may interfere with the responses of the vertical canal.

At the present time, however, we do not use the Bárány reaction on many occasions. It is a severe strain on a patient ill with intracranial pressure and headache to turn and to douche him. Furthermore, the clinical neurologic signs have usually been sufficient to afford accurate localization, or if not, the injection of air has been considered a more direct means of discovering the position of the tumor.

DR. LEWIS FISHER: I should like to discuss further the 116 proved cases of the tumor of the brain that were studied by the vestibular tests. That series included all cases with available records in the University Hospital that had been studied during the past ten or fifteen years, including our earliest cases. Dr. Grant reported at that time that the vestibular findings and interpretations were correct in 90 per cent of all supratentorial lesions and in 80 per cent of the other types of lesions. It seems to me that any method that proves correct in as high a percentage of cases as those quoted by Dr. Grant is certainly an important and useful method, and is not to be ignored. His lack of great enthusiasm at this time can also be easily understood. Neurosurgeons have recently developed and perfected new methods of their own which frequently make it unnecessary to look for aid through any other method. Since vestibular examinations are more or less complex, one can well understand why they are not employed in cases in which the diagnosis is certain. However, it is well to remember that one of the reasons that neurosurgeons are able to diagnose many of these cases is that by the time a patient is brought to the neurosurgeon the lesion has existed for a considerable time, from six months to several years or even longer. This fact, however, is not true in ordinary neurologic practice. There, as a rule, cases are studied much earlier, frequently at their inception. It is in a general neurologic study of a case that a vestibular examination is of most value, often indicating the presence of a lesion even before the ordinary clinical phenomena manifest themselves.

The whole subject of vestibular examination is still in its infancy, and I do not wonder, therefore, that whenever I hear a discussion on this subject I discover things that are rather new to me. Dr. Hunter's paper was no exception. I am unable to verify his observations in my own experience. Personally, I have always tried to simplify procedures as much as possible, and I like to approach every vestibular case in the following manner: The first problem presenting itself is to determine whether or not the patient has an intracranial lesion. Experience has taught that certain vestibular findings always indicate the presence of a lesion of the brain of some sort. Such a lesion may not be a tumor. Disturbances in the brain of many kinds and sorts may produce abnormal vestibular phenomena. Experience, however, often enables one to tell which types of phenomena indicate a mass lesion and which phenomena indicate other forms of disturbances. Having determined that the patient has an intracranial lesion, the next problem is to place it either supratentorially or infratentorially by: the presence or absence of susceptibility to stimulation of the ear, the duration of vertigo after turning, the character of the movements of the pelvic girdle, the presence or absence of past pointing, the character of spontaneous pointing and the presence or absence of spontaneous nystagmus. All these form a picture that practically always locates the lesion in its relation to the tentorium. Should the lesion be placed supratentorially, I try to note the particular group pattern of the responses and call on my experience with proved cases to try to place the lesion more accurately. Thus, in the course of an examination one should not simply be content with irrigating the ears with water and noting whether a response did or did not appear, but particular care should be taken to observe the type of response produced. For example, in observing the nystagmus after douching, the detection of a dissociation of movements of the eyes in a case without any ocular palsies immediately would suggest that the lesion is centrally located. Again, the degree of the patient's susceptibility to the tests is frequently of localizing value. Patients with suprasellar lesions or lesions of the frontal lobe are extremely sensitive to stimulation, while those with other supratentorial lesions are much less sensitive. In a word, a vestibular examination, to be of service, must be watched with the greatest care. Then, and only then, will the examiner obtain information that is reliable and useful. Let me remind you in closing that no method of examination is perfect, nor should any method be expected to prove 100 per cent correct.

DR. J. W. MCCONNELL: Three of the patients whose cases were reported by Dr. Hunter were in my service at the Philadelphia General Hospital; they

were studied and diagnoses were made previous to the Bárány findings. In all, although the neoplasms were located in widely different sections of the brain, there was one element in common: this was the marked change in the corpus callosum. In one, this structure was reported to be as thin as paper. In another, the pathologic report was of an amount of destruction caused by the fungating tumor and direct involvement of the corpus callosum by the tumor itself. In the third case, the destruction of the anterior portion of the callosum by pressure of the growth is specificially mentioned. A fourth case, not reported by Dr. Hunter, presented clinically lateral jerking movements of the eyes, sometimes rotary movements of the eyeballs and once in a while associated movements of the eyes and head toward the same side. This case showed, among other pathologic findings, a remarkable compression of the callosum to about one third of its original size.

I am proposing not to relate these findings in the callosum to the subject under discussion, but rather to emphasize them as points to think about in conditions of the central nervous system in which the symptomatology of intracranial growths or other processes that disturb the movements of the eyes is being considered.

Dr. Hunter has not attempted to determine any new symptomatology; rather, he has tried to clear the atmosphere as to the relation of the Bárány findings in the form of movements of the eyes to definite localization. All that he has said is grist to the mill, but up to the present time the results of Bárány tests and the conclusions drawn from them leave much to be desired. In one of the cases mentioned in his paper, although the lesion was in the right side of the cerebellum, the right ear responded better than did the left and the condition of the brain that gave the Bárány features of a supratentorial lesion caused other symptoms that, for a time at least, led us to favor a clinical diagnosis of frontal tumor. Again, in another case the diagnosis by Bárány test was, first, a supratentorial lesion; second, that the factor producing a hypersensitive reaction was the cause of the nystagmus; yet this patient presented, at autopsy, such tremendous destruction of the cerebrum that it was almost impossible, post mortem, to relate the Bárány findings to any specific changes. In fact, Dr. Hunter says of this case that these reactions might be found in food poisoning, migraine or some foci of infection. The case of the third patient in my service was clinically diagnosed as one of supratentorial lesion, probably of the left frontal lobe. The supratentorial location was emphasized by the Bárány studies, but without definite localization in one or the other frontal lobe.

In reviewing the study that Dr. Hunter has made and the special ocular conditions to which I have not called attention, my thought is that there is much left along these lines for investigation, and I fully agree with him when he says that "a great part of our opinions is conjectural."

DR. R. J. HUNTER: I did not select the cases, but simply took them in rotation. I did not write a statistical paper, and in one of the seven cases did not give an opinion. That happened to be a case of diffuse metastasis. In five others I correctly localized the lesion. I called attention to the one case diagnosed incorrectly. I was interested to hear agreement that the Bárány reaction requires experience in its execution and interpretation. I am convinced that it does have some value in diagnosis. I was hoping that something would be said about conjugate deviation, but most of the discussion has been along vestibular lines. I should have liked to hear what you thought of my statement about the possibility that conjugate deviation might be produced by lesions adjacent to the centers for deviation of the eyes. I did not discuss fully all the findings, such as the normal duration of vertigo. I have not observed this as faithfully as Dr. Fisher has. The class of patients at the Philadelphia General Hospital probably requires more patience in the application of this test than those from a higher walk of life; it is subjective and requires repeated examination to arrive at any opinion of value. The objective tests can be noted no matter what the condition of the patient. Vertigo is something that may vary with the personality and mentality of the patient.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 1, 1931

S. PHILIP GOODHART, M.D., *President, in the Chair*

THE PATHWAY OF VAGUS IMPULSES TO THE CEREBRAL VESSELS. DR. STANLEY COBB, Harvard University.

This is a preliminary report of experiments being carried on by Dr. Jacob Finesinger and myself in collaboration with Dr. Wilder Penfield and Dr. G. Chorobski. The frequently repeated observation of Forbes and Wolff that stimulation of the sympathetic nerve in the neck caused vasoconstriction of the pial vessels over the cerebral cortex and that similar stimulation of the vagus nerve caused vasodilatation aroused our curiosity as to the route by which the vagus impulses reached these cerebral vessels. It is well known that the sympathetic nerves follow the carotid arteries. A series of experiments on cats and monkeys was therefore projected to follow the course of the impulses set up by electrical stimulation of the vagus nerve in the neck.

When the simple procedure of stimulation of the vagus nerve in the neck was carried out with the Forbes' window in the skull over the ipsilateral cerebral hemisphere, microscopic observation of the pial vessels showed that out of 128 trials dilatation occurred 101 times and no change 27 times. With stimulation of the left vagus in the neck and microscopic observation on the pial vessels of the right hemisphere, 29 trials gave vasodilatation 22 times and no change 7 times. Similar experiments with the sympathetic showed that it had a unilateral and ipsilateral distribution; the impulses arising from vagus stimulation evidently are bilaterally distributed to the cerebral hemispheres. Since there are no branches of the vagus between its exit from the medulla and the point where it was stimulated in the neck, and since stimulation of the peripheral part of the vagus, after cutting it close to the medulla, had no effect on the cerebral vessels observed, it was believed that the impulses must travel centrally along the vagus to the medulla, and leave the medulla in some other nerve or tract.

Operations were therefore devised to expose the medulla and allow stimulation of the cranial nerves while observing the pial vessels with a microscope. Stimulation of the spinal accessory nerve caused no change in vessel diameter; likewise the glossopharyngeal, the auditory, the trigeminal and the oculomotor nerves were repeatedly stimulated with no resulting vasodilatation. When the electrodes were placed on the facial nerve, however, a marked increase in diameter of the pial vessels was observed under the microscope (out of 61 trials, dilatation occurred 54 times and no change 7). This indicated that the efferent path had been located in the facial nerve. Further experiments on this nerve were performed to see if the impulses traveled along its whole length: Stimuli applied to the seventh root near the medulla caused vasodilatation under the window; this ceased if the nerve was cut at any point between the electrode and the geniculate ganglion. Cutting the nerve peripheral to the geniculate ganglion caused no interference with the vasodilatation. Stimulation of the seventh nerve peripheral to the geniculate ganglion caused no dilatation. Stimulation at the geniculate ganglion caused the vasodilator response even when the nerve was cut central to the ganglion.

It is known that there is a sympathetic nerve connection between the geniculate ganglion and the vessels of the meninges. Some authors trace this route via the otic ganglion to the middle meningeal artery; others via the superficial petrosal nerves to the internal carotid artery. Chorobski, in an unpublished work, has recently demonstrated a direct anatomic pathway that leaves the facial nerve in the vicinity of the geniculate ganglion and passes to the carotid artery. Penfield's recent description of nerve fibers on intracerebral blood vessels completes the anatomic demonstration.

It seems, therefore, that vasodilator impulses travel up one vagus nerve in the neck, enter the medulla, go to both facial nuclei, thence out along the facial nerve as far as the geniculate ganglion, from which vegetative fibers leave the larger nerve and go to the carotid artery. After reaching the carotid artery, they probably spread out and followed the branching of the arterial tree in the same manner as the vasoconstrictor sympathetic fibers which join the carotid at a lower level.

EXPERIMENTAL MULTIPLE SCLEROSIS IN DOGS FROM INJECTION OF TETANUS TOXIN. DR. TRACY J. PUTNAM, Harvard University.

In 1897, Claude (*Arch. de physiol.* **29**:843, 1897) observed that one of several dogs inoculated repeatedly with minimal doses of tetanus toxin developed ataxia and spasticity and died in about two months. At autopsy, disseminated islands of sclerotic tissue were found scattered throughout the central nervous system, which consisted of a cellular exudate of both vascular and glial origin and demyelinization.

Ceni and Besta (*Riv. sper. di freniat.* **31**:125, 1905) described similar lesions in an animal inoculated intraperitoneally with Aspergillus fumigatus. The animal died three months after the onset of symptoms.

Perdrau and Pugh (*J. Path. & Bact.* **33**:79, 1930) also found similar patches of demyelinization in four of fourteen dogs suffering from the "nervous form of distemper." Three other dogs had areas of perivascular infiltration without loss of myelin. In all three animals, the lesions resembled those described as "acute multiple sclerosis," disseminated encephalomyelitis and disseminated degenerative softening in man. Actual fibrous glial scars have not been observed; hence the term "sclerosis" is probably a misnomer.

For the last three years, a systematic attempt has been made in this laboratory to reproduce Claude's and Ceni and Besta's experiments. No nerve lesions have been found in dogs inoculated with Aspergillus spores or extract. Of about eighty dogs inoculated with doses of tetanus toxin, comparable to those used by Claude, two have shown disseminated encephalomyelitis at autopsy.

Protocols of the experiments are as follows:

Dog 9.—The dog was an airedale mongrel bitch, fully grown.

Feb. 21, 1929: Tetanus toxin, 0.15 cc., was given intraperitoneally.

February 23: Distinct ataxia was noted when the dog attempted to walk.

February 25: The ataxia increased; rhythmic tremor of the right hind leg was noted, especially on voluntary motion.

February 28: The dog was extremely unsteady. Tremor extended to the right front leg. Tetanus toxin, 0.15 cc. was given intraperitoneally.

March 7: The rhythmic tremor increased; 0.15 cc. of tetanus toxin was given intraperitoneally.

March 25: The animal was worse, being unable to stand.

March 28: The dog was moribund. Cistern puncture was performed and death occurred a few minutes later. The spinal fluid showed: 50 lymphocytes; 1 monocyte; total protein, 21; colloidal gold curve, 0123334210. The brain and cord were grossly normal. Sections showed numerous small irregular areas of perivascular infiltration, often accompanied by local degeneration of myelin. The axons were very slightly affected, and no secondary degeneration was seen. The perivascular exudate was made up of lymphocytic and glial cells in about equal numbers. A few rod cells were seen. No striking increase in glia fibers could be demonstrated. There were mild regressive changes in the nerve cells. Small amounts of fat were found. There was a large plaque of demyelinization in the optic chiasm, with cellular infiltration and local meningitis about it.

Dog 37.—The dog was a small white terrier bitch.

Nov. 18, 1929: Tetanus toxin, 0.15 cc., was given intraperitoneally; 10 cc. of spinal fluid was removed by cistern puncture, under negative pressure.

November 19: Six cubic centimeters of spinal fluid was removed from the cistern under negative pressure.

November 25: Tetanus toxin, 0.1 cc., was given intraperitoneally; 1 cc. of spinal fluid was withdrawn from the cistern.

December 15: For the first time, the animal was observed to be slightly unsteady.

Jan. 3, 1930: There was very definite increasing ataxia.

January 15: The ataxia was now at its maximum. The dog constantly fell when trying to run. There was no change in the optic disks.

February 4: The ataxia improved. The spinal fluid showed: 9 cells; total protein, 28; colloidal gold curve, 1123320000. Some of the spinal fluid was inoculated into dogs and rabbits, which showed no symptoms.

April 2: The animal was steadily improving. The spinal fluid showed: 8 cells; total protein, 15; colloidal gold curve, 1233210000.

June 13: The dog was practically well. The spinal fluid showed: no cells; total protein, 49; colloidal gold curve, 1100000000.

December 19: There was no apparent abnormality. The dog was fat and lively. The spinal fluid showed: no cells; total protein, 22; colloidal gold curve, 0012211100. The dog was killed with chloroform.

Autopsy: The brain and cord appeared grossly normal. The other organs were normal grossly and microscopically. Sections of the brain and cord showed rare small lesions, similar in nature to those seen in dog 8, but older, smaller and less intense. Lymphocytes were rare, but there was definite increase in perivascular fibrous astrocytes in these areas.

Five lesions were found in the cord and brain stem in which there were either definite proliferation of fibrous glia or myelin destruction, or both. Myelin breakdown could be followed through the stages of swelling, droplet formation and complete loss. In a longitudinal section, this process might be seen in an isolated section of a fiber that appeared normal elsewhere. The axis cylinders were swollen and twisted, but few were destroyed. Small amounts of fat were found. There was some perivascular infiltration in the optic chiasm and tract, but no actual loss of myelin.

The histologic changes appeared to be identical with those occurring in mild, early, human cases of multiple sclerosis.

THE INFLUENCE OF FLUID INTAKE ON THE INTRACRANIAL PRESSURE IN MAN. DR. FRANK FREMONT-SMITH, Harvard University. (A preliminary report of work being done in conjunction with Drs. W. G. Lennox and H. H. Merritt.)

In his paper "The Mechanical Theory of Epilepsy" (*Am. J. Psychiat.* **8**:783, 1929) and in other papers (*J. A. M. A.* **94**:245 [Jan. 25] 1930 and *J. Nerv. & Ment. Dis.* **71**:481, 1930), Fay stated that pathologic lesions in the arachnoid villi or their failure to develop may lead to delay in the absorption of cerebrospinal fluid. He believed that such delay leads to local accumulations of fluid over the motor cortex, producing local "supracortical pressure," which results in local cortical atrophy and convulsions. He further stated that water drinking may precipitate convulsions in epileptic persons by raising the intracranial pressure, and that fluid restriction prevents convulsions by lowering the intracranial pressure. It is not our purpose to criticize Fay's therapeutic method. We believe, however, that his conception of the mechanics of intracranial pressure is fundamentally unsound, and that whatever influence the level of fluid intake has on the incidence of convulsions does not result from variations in intracranial pressure.

The fallacy in Fay's argument, as we have pointed out previously (The Convulsive State, Baltimore, Williams & Wilkins Company, 1931, p. 617), lies in the assumption that interference with the absorption of cerebrospinal fluid at the arachnoid villi can lead to a higher pressure in the subarachnoid space than in the

ventricles. This is not possible, because as long as any cerebrospinal fluid is circulating from ventricles to subarachnoid space the pressure in the ventricles must exceed that in the subarachnoid space, whether under normal conditions or in the presence of increased intracranial pressure from any cause. Fluid can flow only from a point of higher to one of lower pressure. If we follow what must happen when the arachnoid villi become gradually obstructed, the mechanism will become clear. As soon as the obstruction causes delay in absorption of cerebrospinal fluid there will be a rise in pressure, not only in the subarachnoid space at the point of obstruction but also in the ventricles where the fluid is being continuously formed.

Thus the fall in pressure gradient from ventricles to subarachnoid space will be maintained. The mechanism by which cerebrospinal fluid continues to be filtered into the ventricles from the choroid plexuses against such increasing pressure as the result of partial compression of the choroid plexus veins has already been pointed out (Fremont-Smith, F.: The Nature of the Cerebrospinal Fluid, ARCH. NEUROL. & PSYCHIAT. **17**:317 [March] 1927). As the obstruction to cerebrospinal fluid absorption gradually becomes complete, the pressure becomes higher and higher, and the pressure gradient from ventricles to subarachnoid space becomes flatter and flatter. When at last the absorption of fluid ceases altogether, the circulation of the fluid ceases and the pressure becomes equal throughout the ventriculosubarachnoid system. There is no point in this process when the pressure in the subarachnoid space could exceed that in the ventricles.

The garden hose provides a simple analogy. If one steps on and partially compresses the hose near the nozzle, thus decreasing the outflow at the nozzle, there will be an increase in pressure within the hose not only just behind the point of obstruction but all the way back to the faucet. This increase in pressure will be clearly indicated by the spurting of any leaks in the hose. As long as any water continues to flow through the hose there must be a fall in pressure gradient from faucet to nozzle. If the compression stops the flow altogether, the pressure will become equal throughout the system behind the obstruction. It is not conceivable that such obstruction could result in a higher pressure at the point of obstruction than at the nozzle, for this would reverse the pressure gradient and the water would flow backward up the hose.

Since the pressure is always greater within the ventricles, the subarachnoid space tends to become obliterated, for the higher intraventricular pressure forces the convolutions against the dura and skull. This explains the well known fact that hydrocephalus associated with increased intracranial pressure from whatever cause is almost invariably an internal hydrocephalus. It also explains why the numerous attempts on the part of surgeons to convert internal hydrocephalus into external hydrocephalus were doomed to failure. The higher intraventricular pressure always asserts itself and forces the cortex out against the skull. External hydrocephalus, on the other hand, occurs whenever there is atrophy of the cerebral cortex, the cerebrospinal fluid simply filling the space where brain tissue has disappeared as was pointed out by Dandy (*Bull. Johns Hopkins Hosp.* **34**:245, 1923).

It is evident, then, that Fay's theory of cortical accumulations of fluid (external hydrocephalus) exerting hydraulic pressure on the motor cortex with the production of local atrophy is mechanically unsound. It is nevertheless of interest to study the response of cerebrospinal fluid pressure to water drinking in epileptic persons, because if for any reason they could not excrete water normally, a retention within the organism might lead to increased intracranial pressure.

In a series of tests of water excretion on sixteen epileptic persons following the intake by mouth of from 1 to 2.5 liters of water during a period of from one to three hours, a definite delay in excretion was found in only three. In fact, the diuretic response of normal persons and of most epileptic patients to water drinking is so prompt that often the volume of urine excreted exceeds the volume of water ingested, and the water drinking actually leads to dehydration. Two patients had a convulsion shortly after the test. One of these had shown retention

of water. The other had had a normal diuresis. These results indicate that most epileptic persons have no difficulty in excreting water promptly.

In five epileptic patients we have measured intracranial pressure by means of a water manometer attached to the lumbar puncture needle during such water drinking tests. From 1 to 2.5 liters of water were ingested during a period of about one hour. The spinal fluid pressure was continuously measured during this period and for the subsequent two hours. No rise in pressure took place. Moreover, before the termination of the test the patients had excreted all or nearly all the water ingested, hence no delayed rise in spinal fluid pressure would be expected. When, however, diuresis is inhibited by an injection of pitressin and 2 liters of water is given, a moderate rise of cerebrospinal fluid pressure may occur. Pitressin alone produces little or no rise in cerebrospinal fluid pressure. Fremont-Smith, Putnam and Cobb (ARCH. NEUROL. & PSYCHIAT. **23**:219 [Feb.] 1930) have demonstrated that when water diuresis is inhibited by pitressin, the blood plasma becomes hypotonic (analogous to Weed's experiments of injecting distilled water intravenously). The rise in spinal fluid pressure observed in these experiments is what one would expect, due partly to the osmotic effect and partly to increased blood volume and consequent increase in venous pressure. Even in these cases the rise in pressure is not as marked as occurs every time a person coughs or strains slightly, and can hardly be considered the cause of a convulsion.

McQuarrie and Peeler (*J. Clin. Investigation* **10**:915, 1931) have precipitated convulsions in patients by forcing fluids during the period of the antidiuretic action of pitressin. In one of our patients two convulsions occurred shortly after such a test. The spinal fluid pressure, which had increased moderately during the test, had been lowered to below normal by removal of a considerable quantity of cerebrospinal fluid about one hour before the first convulsion. Another patient had a convulsion after a water drinking test without pitressin. No rise in spinal fluid pressure had occurred during the test, and all the water ingested had been eliminated in the urine before the convulsion happened. It is evident that in these two cases, whatever the relation to the water intake, these convulsions were not precipitated by increased intracranial pressure.

It was thought, however, that such acute experiments hardly give a fair estimate of the effect of longer restrictions of fluid intake. We have, therefore, compared the spinal fluid pressure in two epileptic persons, after a period of normal fluid intake, after a period of marked fluid restriction (but without catharsis or diuretics to aid the dehydration) and after a period of forcing fluids up to from 3 to 5 liters per day. The lumbar puncture at the end of each period was done before breakfast under as nearly identical conditions as possible. There was no significant difference in the pressures under these three conditions. If anything, the cerebrospinal fluid pressure was a little lower after the period of forcing fluids than it was after the period of restricted fluid intake.

Active catharsis or the use of diuretics, we believe, lowers intracranial pressure, but that is a very different matter from varying the level of water intake.

Summary.—These experiments indicate that in the presence of normal kidneys the level of fluid intake, within ordinary clinical limits, has no significant effect on the cerebrospinal fluid pressure in man.

One of the most important functions of the kidneys is to keep constant not only the composition and volume, but also the pressure of the internal environment. This constancy of the internal environment, in spite of well marked variations in the level of water intake, is clearly illustrated by the constancy of the cerebrospinal fluid pressure in these experiments.

DISCUSSION

DR. BERNARD SACHS: Dr. Cobb's exposition of his work was admirable.

In regard to Dr. Putnam's contribution, and in full appreciation of the work that he has done, the great difficulty that has arisen in regard to the entire question of multiple sclerosis has always been: What do we mean clinically when we speak

of multiple sclerosis? I wonder if we should have labeled the case of the dog as one of multiple sclerosis if we did not know that you were trying experimentally to reproduce this disease. If I were looking at that dog impartially, it would have impressed me as having a flaccid paralysis of the hind legs. I did not see much in its clinical behavior that would have suggested multiple sclerosis in man. At the same time, I am impressed with the probability that this diffuse encephalomyelitis, as the sections evidently showed, was the result of a toxic poisoning. The only point that I make is whether the issue is not sometimes confused by trying to construe this as the reproduction of multiple sclerosis, granting the fact that this disease in man implies a very broad conception. I do not suppose that any two clinicians will agree altogether as to whether certain cases are to be defined as multiple sclerosis or not. This is not finding fault with Dr. Putnam's work, but is an attempt to view these animals clinically, with the clinical insight obtained from the study of the disease in man.

What Dr. Fremont-Smith said about the effect of fluid intake on intracranial pressure was extremely interesting. I hope it is not going to lead to an extreme dehydration in the treatment for epilepsy and other conditions which we think may be due to an increase in intracranial pressure.

Dr. Henry Alsop Riley: I wish not only to express my appreciation of these presentations but, in connection with Dr. Cobb's paper, to refer also to some remarks that I made about a month ago in regard to Dr. Fulton's extensive operative procedures on the nervous system. I still feel doubtful about the immediate interpretative applications of extensive mutilating operations on the central nervous system, although I appreciate the necessity for them and the hope that the accumulation of data may in the future supply an answer to some of the riddles of the physiology of the central nervous system. The type of work in which Dr. Cobb has engaged is of quite a different character. It is logical analytical work, which one can grasp perfectly. One sees its rationale easily, and understands readily the conclusions to which the experiments lead.

In regard to Dr. Putnam's contribution, I doubt whether such sclerotic lesions in the nervous system constitute multiple sclerosis. That is a question that cannot be settled by experiments of this kind. In the actual lesions shown, it seems to me that the pathologic process in the spinal cord and in the brain is too similar to that produced by diseases spreading through the vascular system. It differs from that with which I am familiar in multiple sclerosis, principally in the wedge-shaped appearance of the lesions, to which Dr. Putnam drew attention, the widest part at the periphery diminishing to a point as deeper portions of the axis are approached. The lesions seem too distinctly related to the blood vessels, and in the lesions themselves it seems to me that there is a good deal more destruction of the axis cylinder than is usually present in the lesions of multiple sclerosis.

Of Dr. Fremont-Smith's paper I have no discussion, because the presentation was so clear and logical and it was evident from the figures that there can be no differing with the facts that he has presented. Personally, I was glad to hear his paper, because I have never been convinced at all of the dehydration theories expounded by Temple Fay. In a very limited experience with the treatment of patients suffering from convulsions, I have never had any successful result in the control of the seizures by limiting the fluid intake.

Dr. J. B. Globus: I wish to say something about multiple sclerosis as produced experimentally by Dr. Putnam. I think the material presented was very instructive, particularly at a time when many are struggling with the identification of the causative factor in multiple sclerosis—when every one is trying to establish the existence or nonexistence of a relationship between multiple sclerosis and disseminated encephalomyelitis.. I agree that there are some weak points in the pathology presented; for instance, the amount of axis cylinder destruction was much more extensive than is usually seen in well defined cases of multiple sclerosis. And then again, the areas of mesodermal response, which were few and scattered here and there, are very likely purely reparative phenomena. Dr. Putnam did not

make a clear case in favor of the sclerotic character of the lesion, nor did he demonstrate the identity of his experimental lesion with the alterations in encephalomyelitis. Nevertheless, he revealed the highly suggestive results following the intraperitoneal injection of a toxin—the production of lesions of some type in the cerebrospinal system. How the toxin got there is still a mystery, but the fact is clear that the cerebrospinal axis was attacked in a peculiar fashion to produce a dissemination of small sclerotic (?) patches. There was a striking similarity to the lesion of multiple sclerosis in the configuration, in the destruction of myelin and in the washing out of the landmarks at such points, although the areas cannot be identified as truly sclerotic. Of great significance was the fact that in both dogs the optic chiasm was attacked. The optic chiasm is frequently attacked in multiple sclerosis, and that it was involved in the experimental animals cannot be passed over lightly. It is true that Dr. Putnam has not produced undisputed instances of the disease as it is known in man. I do not think that he wished to stress the clinical features or the localization of the lesion—for what is typical in man may not be typical in the dog. This approach to the solution of the problem ought to be followed up, because from the purely anatomic or neuropathologic study of multiple sclerosis not much will be accomplished. The experimental method may open the way, and every attempt in this direction deserves support.

DR. E. D. FRIEDMAN: I was much interested in Dr. Fremont-Smith's findings, because in our work on encephalography we have found clinical confirmation of most of the observations he has made. We have never encountered a case of epilepsy with a striking increase in the intrathecal pressure.

Dr. Fay takes his x-ray plates with the patients in the upright position; one can therefore see why under such conditions collections of air accumulate at the vertex, which is the highest point of the skull. But they do not occur there alone. We have shown that in many cases of epilepsy there are definite evidences of internal and external hydrocephalus. This, we believe, is the result of repeated angiospastic crises, which ultimately lead to ischemic necrosis of brain tissue. The cerebrospinal fluid accumulates in those spaces that are rendered potential vacuums. Any one who has done spinal taps in epileptic persons knows that the spinal fluid usually gushes out in large quantities. We often obtain from 175 to 200 cc. of fluid with ease, and yet it is under normal pressure. This is simply further evidence of the increase in size of the fluid-containing spaces.

DR. WALTER M. KRAUS: It is interesting that Dr. Cobb has demonstrated that the efferent fibers that dilate the pial vessels are limited to the seventh nerve.

In regard to Dr. Putnam's work, may I say that there is a group of cases in clinical neurology in which amyotrophic paralysis results after the administration of tetanus antitoxin? To my knowledge the first cases were reported in 1919 by Lhermitte in Paris. I have recently observed a case. A young man was riding on horseback and was thrown. He had a severe abrasion of the shoulder. He was given tetanus antitoxin in the opposite arm. Within ten days he had severe neuralgia in the arm in which the antitoxin was given, followed by difficulty in the use of the shoulder. He has progressed to a stage where he has muscular atrophy in both shoulders and arms. This is unquestionably progressing rapidly. There is no evidence of any other origin for the disease in this man. I wonder whether Dr. Putnam may not have provoked a similar reaction to tetanus toxin in dogs, which may reproduce a similar condition to that which we know occurs very rarely in man after tetanus antitoxin is given.

DR. LAWRENCE S. KUBIE: Multiple sclerosis is a name for the clinical result of a process in the nervous system. This process consists first of patchy demyelinization, followed by slow scar-tissue formation of varying degrees. This process can undergo spontaneous remission and exacerbation. We do not know what agencies start this process, nor whether it can be produced only by one set of factors or by more than one. In all respects except one, Dr. Putnam produced in the nervous system of two dogs a process identical with the one described. The only point in which his disease in dogs does not exactly imitate the disease

in man is in the absence of spontaneous remission and exacerbation. Nevertheless, it seems to me that we are unable to say whether or not this experimentally produced disease is identical with multiple sclerosis for the obvious reason that we do not know whether multiple sclerosis is one disease or a group of diseases. One of the reasons for our difficulty is the fact that patients with multiple sclerosis almost never die in the acute stage. It is therefore not possible to hope that we could demonstrate in the brain tissue of chronic cases any active virus or infection. For this reason I regret that Dr. Putnam did not make direct inoculations of bits of brain tissue from his experimental animals into the brains of other animals, with the technic that is customarily used in the study of virus infection of the central nervous system. It is true, of course, that the pathologic picture he has produced does not resemble the pathologic picture of ordinary virus diseases. Nevertheless, our knowledge of these diseases is still so limited that I should hesitate to rule out the possibility of a virus process solely on the basis of the pathologic picture. Certainly, it is hard to understand why only two dogs out of so many responded to Dr. Putnam's experimental procedure and developed this disease, unless his experimental procedure activated something in the dog which later produced the disease.

His observations also remind us that in some of the early French literature there are reports of cases of multiple sclerosis which developed acutely after typhoid fever. In that connection I know of a case in which the disease seemed to come on after inoculation against typhoid fever.

With regard to the paper of Dr. Fremont-Smith, I have only one question to raise. The differences in levels of pressure between the arterial and venous ends of the capillaries, the cerebrospinal fluid and the whole venous system are very slight. Is it not conceivable, therefore, that even slight shifts up or down in the general levels of pressure in the cerebrospinal fluid spaces might affect the freedom of vascular flow? If this is true, we can picture cortical atrophy resulting from even moderate increases in cerebrospinal fluid pressure, even when this increase is diffusely distributed throughout the ventricular and subarachnoid system. We know that the cells of the cortex are among those that are first to be damaged by slight reductions in blood supply. Local differences in vascular supply might make the cells of one area more susceptible than those of another area when slight changes in pressure have occurred. If true, this may partially reconcile the differences between the work of Drs. Fremont-Smith and Fay.

DR. M. NEUSTAEDTER: My experience in encephalography is analogous to that of Dr. Friedman in that in every case there was a gush of spinal fluid, yet the pressure never exceeded 12. It usually lies between 8 and 12 mm. of mercury. I always make encephalographies and lumbar punctures in the horizontal position, and photograph the patient first in the horizontal and then in the vertical position. In only a few cases was there an internal hydrocephalus.

DR. RICHARD M. BRICKNER: Concerning Dr. Kubie's point that a latent virus might have been activated by the injected toxin, that is possible but not probable. The chances are that the lesions in these dogs were caused either by the toxin itself or by some change produced by it in the internal economy. If this is true, then a toxin can be responsible for disseminated lesions in the nervous system in which myelin is lost. Multiple sclerosis itself may also be produced by a toxin.

I think it is perhaps going a bit far afield to try to establish a direct relationship between the lesions produced in these animals and the disease multiple sclerosis in man. I believe that the final evidence regarding the cause of that disease will come through the production of the disease in animals by some product of patients with multiple sclerosis, and not by any indirect method. After that step has been accomplished, an analysis of the substance which produced the lesions might well show what the exact nature of the agent is.

The work of Dr. Putnam is very important, not because he has definitely produced human multiple sclerosis in animals, but because he has shown that, in all probability, a condition at least resembling it pathologically can be caused by the activity of a nonliving agent.

DR. C. BURNS CRAIG: I wish to ask Dr. Cobb a question. Since it has been declared by some that the seizure in epilepsy is due to vascular spasm in the cortex, did he produce convulsions in the cat during the time of the stimulation of the sympathetic, when he was undoubtedly causing vascular constriction over a large portion of at least one hemisphere?

DR. STANLEY COBB: All the animals were under amytal and therefore had no convulsions. We have done some experiments on animals without ether, using local anesthesia; with this method we have occasionally obtained convulsions accompanied by visible vasoconstriction. It is difficult because one has to use local anesthesia, watch the animals closely and avoid hurting them.

DR. TRACY J. PUTNAM: I am well aware that this paper does not solve all the problems of multiple sclerosis. It is true that neither of my animals complained of scanning speech, nystagmus or intention tremor, and it would have been most difficult to make the diagnosis clinically if it had not been for the cases of Claude and Ceni and Besta. However, we know that human multiple sclerosis is a protean disease. I have seen human cases with rhythmic hemitremor, and many with ataxia and paraplegia as the only manifestations. We know that the physiologic response to a given injury of the nervous system of a dog is much less than that to a corresponding injury in man. Moreover, compensation and recovery are more rapid in animals.

In any case, the final diagnosis of multiple sclerosis in human beings, and I should think also in dogs, rests on the pathologic anatomy of the disease. Irrespective of symptoms and course, if typical disseminated plaques are found at autopsy, we must call the disease multiple sclerosis. As to whether the lesions here demonstrated can be considered typical or not, I can add little to what the lantern slides show.

Should the lesions be considered inflammatory? I do not know. Lymphocytes and plasma cells occurred in the exudate, as they do in so-called acute encephalomyelitis. They also occur in unmistakable cases of multiple sclerosis, in much the same proportion as in the slides from these animals, as I have been able to convince myself from human material in our collection. Similar exudates may be produced by bland emboli and thromboses.

Of course, the large dense glial scars seen in human cases could hardly be expected in plaques a year old in a small animal. As to the persistence of axons, this is a relative matter even in human multiple sclerosis and, according to Hassin and Dawson, probably a large proportion are lost, but the remainder are packed so densely together in old lesions that the loss is not obvious. Here again, the lesions in my dog should be compared with early and small human lesions.

That the myelin destruction is chiefly perivascular constitutes a similarity with human cases, rather than a point of difference. The perivascular distribution of small plaques in human cases has been described by many observers, and the matter appears to be settled by the work of Dawson and of Falkiewicz, who independently reconstructed lesions and found that they followed vessels for some distance. Larger lesions doubtless form by coalescence, and this tendency may be seen in the dogs' cords also.

In fine, I do not hold that these animals reproduce all the details found in all human plaques, but rather the essential features of human lesions of corresponding size and age. It seems to me difficult to make any other diagnosis than that of sclerotic plaques. The lesions are not focal necroses; they are not systemic degenerations; they are not multiple abscesses, and so on.

As to the relationship between the toxin and the lesions, I am at a loss. A virus may be the missing factor, yet the lesions do not in the least resemble those of rabies, herpes or distemper, the only known virus diseases affecting the dog's nervous system. The absence of epidemic features is also against the presumption of a virus.

In regard to Dr. Kraus' interesting case, this is particularly suggestive because a case of multiple sclerosis following the administration of tetanus antitoxin has been reported by de Massary and Mevel (*Rev. neurol.* **1**:347, 1926) and a case

of myelitis following typhoid inoculation by Divey, Moreau and Ory (*J. de neurol.* **29**:369, 1929). We tried to produce lesions with antitoxin, and also with incompletely neutralized toxin-antitoxin mixtures, but without success.

DR. FRANK FREMONT-SMITH: Dr. Sachs asked whether this was going to lead to prolonged, severe dehydration therapy. I really do not feel qualified to comment on the therapy, because I have not had sufficient experience. There is more to learn about the relationship between water-intake and convulsions. The only point I wish to make is that if convulsions are inhibited by restricting fluids, this is not brought about through lowering the intracranial pressure.

I am interested in what Dr. Friedman had to say. It may explain why so much cortical atrophy may be found in some cases of epilepsy, i. e., because the patients have the encephalograms taken in the upright position.

I agree with Dr. Neustaedter that cortical atrophy or internal hydrocephalus does not occur in every case of epilepsy.

Dr. Kubie brought up an interesting point. He is right that with increased intracranial pressure there will be a slowing of the blood flow through the brain, and if that is sufficient to produce anemia of the brain atrophy will follow. One would expect such an atrophy to be a diffuse process. All the gray matter should respond in about the same way, and the white matter in about the same way, which does not fit in with these cases of epilepsy, in which at autopsy we find atrophy of particular convolutions near together. Some are lost, and others are well preserved. This is much more suggestive of a pathologic process beneath those convolutions than it is of a diffuse pressure atrophy. Moreover, the pressures encountered in epilepsy are not high enough to produce cerebral anemia.

Book Reviews

Ueber Apraxie. By Otto Sittig. Price, 24 marks. Pp. 248. Berlin: S. Karger, 1931.

Sittig's monograph is based on clinical study. He divides knowledge of apraxia into the pre-Liepmann and post-Liepmann eras. Hughlings Jackson was apparently the first to describe apraxia, but never gave it a name. In 1870, Finkelnburg gave the name asymbolia to all forms of disturbance in symbol formation and understanding. In 1870, Meynert defined motor asymbolia as the failure to appreciate the use of an object, along with a loss of perception of it. Sensory asymbolia was defined as the loss of recognition of objects. These disorders were looked on by Pick as apraxia, but Heilbronner clarified the problem by calling the motor type apraxia and the sensory agnosia. In 1899, de Buck, a Belgian, published a case of apraxia and preceded Liepmann's views by a year. However, it was not until Liepmann, in 1900, that ideas of apraxia were more or less clarified. He showed that the disturbance was unilateral, in the right hand only, and therefore not an agnosia or aphasia. He pointed out further that only certain motor acts were impossible, such as those that required auditory, visual and tactile senses for their performance. He localized the lesion causing apraxia in the left parietal lobe, and indicated the rôle of the corpus callosum in the syndrome, pointing out that when this was injured there resulted a left-sided apraxia. Liepmann made a classification of apraxia into limb-kinetic, ideokinetic and ideational apraxia. Pick criticized this sharp distinction between motor and sensory apraxia, and later Liepmann came to agree with him. Monakow disagreed heartily with the idea of a localized praxis center, arguing that the process was spread over the entire brain. He pointed out also that apraxia almost never occurs in a pure form, but is usually combined with aphasia. Cases of apraxia have been reported with lesions (1) in the corpus callosum, (2) in the gyrus supramarginalis, (3) in the frontal lobe and (4) deep in the parietal lobe.

Sittig calls attention to the occurrence of trunkal apraxia, which was pronounced in a case that he reports. The patient had great difficulty in lying down, often taking one-half hour or more to do so. Trunkal apraxia is rare; a patient of Zingerle's was unable to sit up owing to apraxia, and a patient of Heilbronner's was unable to figure out how to get into bed; in Kroll's case there was great difficulty in getting up from the prone position, and in a case of Klein's a similar disability was noted. Cases of gait apraxia have also been noted. Trunkal apraxia may occur in cases of limb apraxia, the latter being more prominent than the trunkal disturbance. Sittig reports a case in which the face and arms were but slightly apraxic, the legs were rather markedly so and the trunk was much the most involved.

It is in the teachings of Hughlings Jackson that one obtains the clearest account of apraxia; Sittig uses Jackson's observations to explain some of his own findings. Jackson's studies of hemiplegic patients showed that some parts of the body are more involved in the hemiplegia than others. He showed that in destructive lesions of the brain the more voluntary functions suffer most, the automatic functions least. Furthermore, the more severe the lesion, the more severe the voluntary loss. Sittig uses this to explain the late occurrence of trunkal apraxia, and its presence only in combination with other parts of the body. The trunk muscles are not involved in apraxia for the same reason that they escape in hemiplegia, because their function is automatic and their implication in cortical disease is therefore less. If Jackson's law is correct, then apraxia should occur in those parts of the body with a complicated voluntary function. Sittig tested this law in seventeen cases of apraxia that he studied and found it to hold true. Sittig reports a case of facial apraxia. Furthermore, if Jackson is correct, one

should not observe an isolated apraxia of the arm or leg. Sittig has never observed this in his own material, but Foix reported a case of apraxia in the right hand only. Further study in this case showed, however, that the right hand was chiefly involved, but that the arm and leg were also slightly implicated. There are no cases of isolated leg apraxia in the literature. Sittig reports such a case in his monograph. There was no gait or trunkal apraxia. According to Jackson's law, the relative rarity of isolated apraxia in one part is due to the fact that in the higher centers several bodily parts are represented, the higher the parts, the more body paths being represented. If one tests Jackson's law by studying the occurrence of apraxia in various parts of the body, one finds the parts involved in the following order: hands, face, legs and trunk.

Liepmann pointed out what he called sympathetic apraxia, cases of right hemiplegia with a left-sided apraxia. He thought that they were due to a connection of the right and left arm centers through the callosum. Any supracapsular lesion will produce this effect, and involvement of the callosum alone will cause a left-sided apraxia.

Sittig discusses fully the question of localization of apraxia. It is much in doubt.

The monograph is well arranged and constitutes a thorough review of knowledge of the subject. Its attempt to aline the reported cases with jacksonian principles of cerebral functions is wholly praiseworthy. There is a good review of the psychology of apraxia. The book can be highly recommended. It constitutes an excellent résumé of the subject.

Grundlagen einer neuen Psychiatrie. Ein Lesebuch für Laien, Studenten und Forscher. By Richard Wahle. Price, 4.80 marks. Pp. 87. Vienna: Steyrermühl, 1931.

With assurance that would be praiseworthy if his book offered anything new and correct, the author of this book lays down what he considers "the foundations of a new psychiatry." Of clinical psychiatry as it stands today he thinks very little. For psychoanalysis in its entirety he has nothing but sneers and contempt. "Individual psychology" finds more favor in his eyes. But the modern doctrine of schizophrenia and schizoid personalities he considers "valueless." What is needed, he thinks, is a "true conception, both psychologic and physiologic, of all so-called mental phenomena." This only possible "true conception" is offered by the author. It has been previously described by him, very fully, in a book called "The Origin of Characters." In the present volume, the author frequently admonishes the reader to study this previous book; it is, as he says, so "rich in content" that every time he himself "looks into it he learns something" (p. 74).

Whatever one may think of the author's own psychology and physiology, it certainly has the advantage of great simplicity. There are "motor fields" and "sensory fields." These the author believes to be localized in the cerebral cortex. But he states that it makes no real difference where they are; if future research should show that they are in another part of the brain, his theories would be just as valid. These two fields can become stimulated, inhibited, disordered, and so on. The elementary processes that are thus caused explain everything. "There are . . . no acts of thinking, feeling or volition" (p. 10). All the complex concepts that psychologists use are wrong and misleading. "There are nothing but bundles of representations, primary and secondary, with further primary and secondary excitations of the body and their beginnings" (p. 10). "The thinking process is nothing but a series of individual representations" (p. 11).

This psychology, if it can be so termed, which recognizes "only final elementary events," the author applies in the present volume to the whole field of psychiatry. He discusses melancholia, mania, "so-called special manias" (like kleptomania), amentia, alcoholism, thyreogenic psychoses, paranoia, paranoia querulans, dementia praecox, dementia senilis, dementia paralytica, neurasthenia and hysteria. His

conception of all these different psychoses can be summed up in one sentence: In all of them there is something wrong with the "motor field," the "sensory field" or both.

The author has unusual ideas about the data of clinical psychiatry. He thinks that mania and melancholia are so different from each other that it is "illogical" to speak of manic-depressive psychoses. In mania, the Argyll Robertson sign may occur (p. 26). A patient who confabulates "lies" and "realizes with some pleasure that he is lying" (p. 28). The author believes that psychiatrists in general regard kleptomania, pyromania, etc., as closely related to mania (of manic-depressive psychoses). He states that there is "no reason to see anything psychotic" in the delusions of jealousy of chronic alcoholic patients (p. 43). In delirium tremens, "hallucinations are very rare" (p. 43), but "inflammation of the nerves is frequent" (p. 44). "In dementia there is an almost complete lack of emotions" (p. 66). Persons who suffer in youth from neurasthenia or claustrophobia may develop, later, paralyses and diseases of the spinal cord. It is therefore indicated that "such milder states of nervousness" should be treated with "strong chemico-physical means."

In developing his ideas, the author uses some strange neologisms. Instead of "consequences," he speaks of "subsequences," a term that he introduces specifically (p. 21). He distinguishes between "destruction of the fields" and "disorders of the function of the fields"; the former are "psychoses," and latter "Mankoses."

The book is well made and well printed. The only literature quoted, apart from the author's own writings, is the textbook by Pilcz.

L'intuition délirante. By R. Targowla and J. Dublineau. Price, 35 francs. Pp. 316. Paris: Norbert Maloine, 1931.

The present volume is an amplification of a previous paper by one of the authors (J. Dublineau) for which he received the Esquirol prize in 1929.

By "delusional intuitions" the authors understand a special symptom. It consists of delusional ideas that "come to consciousness brusquely and spontaneously." They seem self-evident to the subject and are recognized by him as entirely "personal ideas." They have no connection with "hallucinations, pseudo-hallucinations, illusions or delusional interpretations." The authors believe this symptom to be a clear entity that can be readily distinguished from other psychopathologic symptoms. Delusional intuition has three essential characteristics: (1) "It is an immediate judgment"; (2) "it impresses itself on the subject with absolute certainty, without any proof"; (3) "it is entirely personal and is not derived from any outside events." The symptom is said also to be "very simply defined" by its negative aspects, as it is neither a delusional interpretation nor a hallucination in the widest sense.

The authors state that although the term "intuition" has been used before, a clear description and clinical study have been lacking. They actually consider that "intuition" is a mechanism fully explaining the origin and development of certain delusions.

Targowla and Dublineau do not attempt a psychologic study in the analytic or dynamic sense of the development of "delusional intuition" in the individual patient. On the contrary, they seem to believe that this intuition is a fundamental process. It arises on the basis of a "psychologic automatism" which in itself is founded on "somatic conditions." Other psychologic data that come up in the individual patient with this symptom belong to an "intellectual superstructure" of delusions.

The greater part of the book is occupied by sixty case histories. They illustrate the various modes of manifestation of "delusional intuition" in different conditions. Delusional intuition is found chiefly in "delusional syndromes developing on a disorder of the affectivity (namely, anxious, hypomanic, schizophrenic or psychasthenic states)," or in association with conditions of confusion. It is especially noted in conditions in which affective and toxic infectious disorders are combined.

There exists, according to the authors, a disposition toward intuitions that is "probably dependent on a certain disequilibrium of the organo-affective tonus" (page 254).

This book has the merits and the faults of the modern French school of psychiatry. The descriptions of symptoms and "states" are subtle and clear, but the whole discussion remains on a formal ground. Any deeper psychologic analysis is shunned, and too much emphasis is given to terms like "psychologic automatism," which say little and explain nothing. After the perusal of a great deal of descriptive material, the reader is finally left with the statement that "Intuition is a manifestation of psychologic automatism."

Kleist's studies of what he calls "acute inspiration psychosis" and Wernicke's discussion of "autochthonous ideas" are not mentioned. The bibliography refers almost exclusively to French authors.

Probleme der klinischen Psychiatrie. By Kurt Schneider. Price, 2.60 marks. Pp. 46. Leipzig: Georg Thieme, 1932.

In this small pamphlet the author reviews some of the main trends of clinical psychiatry in the period covering more or less the last decade. Since the text consists for the most part of abstracts of the literature in question, it does not lend itself to a comprehensive review of its content. Schneider is critical throughout, although he does not always discuss why he accepts or rejects certain contributions. For example, he makes the statement that Kleist's psychopathologic views are for the most part "unacceptable" to him, but he does not indicate their defects. In the same way, he fails to express concretely why he accepts fully such entities as the motility psychosis.

The literature considered is restricted almost entirely to a very special group of psychiatric literature; the content of the book would be more clearly indicated if its title were: "Problems of Academic German Clinical Psychiatry." French psychiatry is not mentioned, even when the context would seem to require it. Equally striking is the omission of any mention of psychoanalytic contributions. For the influence of psychoanalysis on psychiatric thought — and the influence is considerable, even on psychiatrists whom the author fully discusses, e. g., Kretschmer — is certainly one of the chief problems of clinical psychiatry during the period covered by Schneider's book.

In his critical summing up, the fact stands out that the author wishes to make a fundamental distinction between psychoses and reactions of abnormal personalities: there is "a sharp either — or," and the difference is "absolute" (p. 36). It is somewhat confusing that he proposes to replace the term manic-depressive psychosis by cyclothymia, while on the other hand he states that personalities should never be called "cyclothymic." This is in contradiction to current usage. Of hereditary research the author expects little. Heredobiologic study of personalities, especially, he considers is "not possible" (p. 37). He seems to overrate considerably the value of anatomic research for the study of "disease entities." He recommends "pathophysiologic research," but fails to indicate more concretely what topics and methods he has in mind.

The book has the advantage of being written clearly and in a simple and direct style. It is unfortunate that the survey itself is not broader and more constructive.

Let's Be Normal! The Psychologist Comes to His Senses. By Fritz Künkel. Translated by Eleanore Jensen. Price, $3.50. Pp. 290. New York: Ives Washburn, 1929.

This book is by one of the members or followers of the adlerian school of psychology, better known as individual psychology. It has been written for the purpose of concerning itself with the problems of everyday life as seen from the fields of psychotherapy, pedagogy, public welfare and the self-education of

the individual. The author is convinced that the individual psychology of Adler has helped in getting the proper understanding of human character. Described throughout this work is "the process of clarification." "Human character as presented in this book will be understandable only to those who have experienced clarification or been close to it."

Part I discusses the forms of character, dealing largely with the various degrees of egocentricity and other complications if the egocentricity is too extreme for easy adjustment. This keynote of egocentricity goes through the entire volume. Through the process of negative experiences, suffering and making objective most of the experiences in life the process of clarification takes place.

The development of the human being appears as a series of progressive steps toward clarity which his suffering compels him to take. The stages of clarification are outlined as: (1) clear insight; (2) admission; (3) acceptance of reality. Various phases of life from childhood to adolescence and the usual adult experiences are discussed from this point of view. The growth of character is described in detail. It is taken up with a description of the development of objectivity in the child as influenced by the different egocentric persons surrounding the child. There are good implications in this section outlining what technic should be used in training the child into superior performance. Otherwise, there is nothing of particular note in the book. The detailed discussions on various points seem to be too long and involved in proportion to the material covered. The volume presents an interesting point of view of a limited type and has not great content for use in fields with which it might be allied.

Grundriss der Alkoholfrage. By Dr. Rudolf Wlassak. Second edition. Price, 10 marks. Pp. 255. Leipzig: S. Hirzel, 1929.

The author of this work, originally an investigator in physiology, was for many years in charge of the division for alcoholic patients in the large institution Am Steinhof in Vienna. In the present, second, edition of his Grundriss he has left the best presentation of the complex field available so far. After a discussion of the composition and production of alcoholic beverages, he presents the physiologic effects of alcohol (pages 12 to 44) as narcotic and energetic data; then the influence on diseases and deaths (pages 45 to 100), the alcoholic psychoses, alcoholism and progeny and crime, the consumption, the social conditions, the results among the masses and the measures against alcoholism (pages 177 to 234). A valuable bibliography is offered. The book is so condensed and representative in its presentation of a vast amount of material that it is impossible to say more in a brief review than that it is and may easily remain an outstanding model of fairness and objectivity and the groundwork for any new presentation. It would be desirable to have it in a translation with adequate consideration of the special needs and conditions in the United States.

A Text-Book of Psychiatry for Students and Practitioners. By D. K. Henderson, M.D., and R. D. Gillespie, M.D. Third edition. Pp. 595. New York: Oxford University Press, 1932.

The necessity of a third edition of a textbook within a period of five years is a measure of its success. As an evidence of the growth of psychiatry, it has been found necessary to add a chapter on the psychiatry of childhood. The growth of the Child Guidance Clinics initiated by the Commonwealth Fund of New York has made this necessary. Another welcome addition is a résumé of the more important laws governing mental disorders in the United States of America and in continental countries.

Some alterations have been made here and there. In the chapter on epilepsy some new material has been added, but in some regards this chapter would have been better off without some of the newer theories and treatments, notably the theory that epilepsy results from watery edema of the brain and that attacks

can be cured by dehydration. This contention has not as yet had time to prove its merit, and results so far have indicated that neither the theory nor the treatment rests on a firm basis.

Les agents physiques dans le traitement des maladies nerveuses. By H. Schaeffer and E. Biancani. Price, 20 francs. Pp. 187. Paris: Masson & Cie, 1932.

This is a pot-boiler, a compilation of various types of physical therapy and their application in the treatment of nervous disorders. The types of case range from intracranial tumors to Volkmann's contracture and hysteria. No section of the book receives adequate treatment or a critical evaluation of the results. The methods of application of the treatment are discussed too briefly to be of any real value, and the book resolves itself into a compilation of what has been attempted in the way of physical therapy in nervous disorders. Not the least impressive are fifty pages in the back devoted to advertising publications by Masson.

Lebensalter und Geschlechter. By Prof. Dr. C. H. Stratz. Price, 15 marks. Pp. 194, with 21 tables and 84 illustrations. Stuttgart: Ferdinand Enke, 1926.

This last work of the noted student of the body forms of man, and especially of the sexes, offers a simple survey of the growth and involution patterns, the successive periods of life and the influence of the vital processes on the body form, as well as data on the duration of life. The second part (pages 97 to 182) deals specifically with the differences between the sexes.

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